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THE

MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—9TH YEAR. SYDNEY: SATURDAY, OCTOBER 14, 1922. No. 16.

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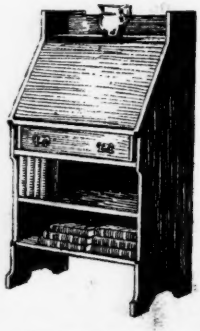

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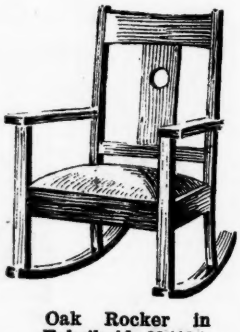

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URINARY INFECTION.[1]

BY A. S. ROE, B.A., M.B., B.CH. (OXON.), F.R.C.S. (EDIN.),
Honorary Urologist and Assistant Surgeon, Mater Misericordiæ Hospital, Brisbane.

SINCE last reading a paper on urinary infection to you I have somewhat altered my idea of the pathology of the condition and shall endeavour to put before you a way of looking on these cases which I venture to think is in many ways original. It is based very largely on clinical observation, as the difficulties of making exact scientific investigations amongst private patients are colossal.

There are several factors concerned in the production of urinary infection, which may be roughly classed as follows:

(1) Absorption of bacteria into the blood from some focus elsewhere in the body, the commonest lesions being in the bowel, prostate, teeth, tonsil and nasal cavities.

(2) Obstruction to the urinary outflow, *e.g.*, ureteral stenosis or stone, enlarged or inflamed prostate, stricture of the urethra, pregnant uterus or any other pelvic tumour.

(3) Disturbances in metabolism, which are connected with the production of a highly irritating urine. It is on this subject I intend to expend most of the time at my disposal to-night; for the theory I accept sole responsibility.

These three classes may operate singly or in conjunction and it is my belief that class (3) from the point of view of preventive medicine is the most important. Accepting Crabtree's theory of the hæmatogenous origin of renal infection, I was led to look on the kidneys as the main means of elimination of all bacterial contents of the blood and further than that of all the chemical irritants in the blood. Then two highly significant clinical facts struck me. The first is that a definite acid condition of the urine is often the most striking feature of the case and the second that fatigue in the majority of cases is the most suggestive factor in determining the attack, whether the case is a chronic or an acute one. Then I remembered a very interesting experiment in physiology, by which it was proved that fatigue in muscles was caused by incomplete oxidation of tissue katabolism, that these incomplete products were of a highly acid nature, being mainly sarcolactic and lactic acid, instead of CO_2 and H_2O, and that if these products were injected into the arterial circulation of a healthy muscle, the muscle immediately showed signs of fatigue. Also, experiments done on the Oxford University "eight" in training (men representing the pick of England) showed a definite number in whose urine

[1] Read at a meeting of the Queensland Branch of the British Medical Association on September 1, 1922.

blood and albumin appeared, which cleared up completely after rest and apparently could not be accounted for except by blaming the undue fatigue.

Another physiological fact is the extraordinary influence of nervous phenomena on the metabolism of digestive glands. With these established facts in my mind it seemed not unreasonable to argue that disturbances in the metabolism of each and every tissue in the body must be continually taking place, that the products of such incomplete metabolism must be of a highly irritating nature, must be carried in the blood and be eliminated almost completely by the kidneys and in all probability will be highly acid in character. Following this line of thought, I attempted to find some such disturbance in metabolism as a causative agent in the actual incidence of any urinary infection.

In the large majority of cases of bacterial infection of the urine—if you examine the urine of your patients microscopically you will find their name is legion—I believe this disturbance in metabolism produces a highly irritating blood, which eventually not only impairs the kidney's efficiency, but lowers its vitality, making it unable to resist the attack of the multitudinous bacteria, which in the nature of things must at times be passing through them. Two very striking facts have contributed to this view. Firstly, there is the wonderful constitutional improvement caused by flooding the tissues with fluids and alkalies, even when there is no apparent alteration in the number of bacteria and amount of pus in the urine. Secondly, in a very definite number of patients complaining of frequency and painful urination with pain in kidney region, headaches, etc., on whom I had done a cystoscopic examination and found trigonitis and marked ureteritis, the bacteriological report has been "Highly acid urine; urates in excess; nothing else abnormal."

The physiological conditions most likely to cause these disturbances in metabolism are associated with deficiencies in diet, disorders of the internal secretions and physical or mental disturbances which can clinically be classed as fatigue. Lest you should think these classifications are too vague and unpractical, I will endeavour to indicate their application in clinical medicine.

Firstly, as regards diet, we know that starvation produces an acidosis; so, I believe, do all the deficiency diseases, scurvy, rickets, etc.. Recent experiments with feeding healthy men with large amounts of meat showed the appearance in almost every case of blood and albumin in the urine. Now the first two years of life are full of obscure conditions of ill-health. A urinary examination will show in practically every case a highly acid urine, generally swarming with bacteria and often associated with pus. Deficiency in diet is always mentioned as a factor in the production of innumerable cases of nephritis developed on active service during the late war.

In discerning the next class, *viz.*, disorders of the internal secretions, I must necessarily be even more vague, though it is here I believe my theory will prove of most use in preventive medicine.

Pregnancy is naturally what comes to one's mind first. There is no doubt that in pregnancy, especially a first pregnancy, marked changes take place in the character of most important internal secretions, as well as the toxæmia caused by the developing fœtus. There is also no doubt that many pregnant women complain of marked urinary symptoms and that certainly in Brisbane a considerable percentage of pregnant women develope an acidosis or a phosphaturia, practically always with a bacilluria and very often with pus. In quite a number of these women, if a catheter specimen be taken, an acidosis will be discovered after parturition, even if previously treated with alkalies. This may be accounted for both by fatigue, injury to the lower portion of the urinary tract and even a disorder of internal secretion associated with lactation. If a catheter specimen of the urine in such cases be examined after parturition you will be surprised to find how often albuminuria will be present, even if it has not been discovered beforehand. I cannot help forming the opinion that some such condition as I describe must be the precursor of all cases of eclampsia and would urge that more consideration should be given to pregnant women when they recount their tales of urinary distress, indigestion, fatigue, etc. (all signs of renal impairment), and that treatment should not be withheld till great pain, gross pyuria or albuminuria put in an appearance. That early treatment can be of value I have proved absolutely to my own satisfaction. During lactation women are very liable to exacerbations of acidosis, according to my theory due mainly to fatigue and disorders of internal secretions connected with lactation. This is perhaps not quite so vitally important. This condition, if untreated, has an important effect on her powers of nursing the baby and, I believe, is the cause of many cases of early weaning and of all cases in which the new-born babe passes frequent green motions, etc., even when breast fed. In carrying on this theory I am naturally led next to the case of the new-born baby. I do not think it has ever been proved, nor does it appeal to my imagination, that bacteria can pass through the placental circulation from the mother to the baby, but it must be possible for chemical bodies to do so, or at any rate the chemical constitution of the mother's blood, and her powers of elimination must have a vital effect on the chemical contents of the fœtal blood. Consequently in pregnant women with chemical toxæmia and consequent impaired renal function the child is born with a chemical toxæmia and its renal function correspondingly impaired. I venture to suggest this as a reasonable explanation for the high percentage of ill-health and even early deaths in infants born of eclamptic and pre-eclamptic mothers.

I cannot resist telling you briefly of three cases which support this view. The first patient had only one kidney and this had been incised. She had a hopeless chronic pyelonephritis, was practically bed-ridden and unfortunately became pregnant. At the third month she sent for me, when she had started to miscarry. I had her in bed for about four months, suffering all the time with attacks of vomiting, headaches, backache, etc., during which

time she lived almost entirely on morphine, atropine and rectal saline injections. During all that time she only had occasional traces of albumin in her urine. Cæsarean section gave her a live, apparently healthy child, but after a couple of days it rapidly wasted and in a week died of a violent convulsion.

The second patient had had fits with her first baby some eight years previously. She became pregnant and was treated by me from about the third month. She then had a phosphaturia, with bacteria and pus, pain in her side and headaches, tiring very easily. I examined her urine very carefully and up to the eighth month there was no trace of albumin. However, she then had a violent attack of headache, backache, vomiting, etc.. She waited for three days, in spite of my having warned her, and then sent for me. She was then desperately ill and had a considerable amount of albumin in her urine. Cæsarean section revealed a live, small child, apparently otherwise normal. It wasted rapidly, however, and was dead in less than a fortnight.

The third case was less severe. I had treated the patient for chronic pyelonephritis ever since the birth of a dead seven months' fœtus about two years previously. She did well with medical treatment, but became pregnant and almost immediately had violent backaches, headaches, nausea and frequency. Rest in bed gave temporary relief, but about the sixth month fœtal movements ceased, she developed severe headaches and dysuria and a trace of albumin appeared in the urine. Rest, salines and morphine soon relieved her; the fœtal movements returned and she felt remarkably well. With the two previous cases fresh in my memory, I kept her in bed, regulated her diet; in fact, kept her katabolism as low as possible and encouraged her elimination as much as possible. She gave birth to a small, apparently normal child at eight and a half months and, although she is not in perfect health herself, she is nursing the baby, which is doing satisfactorily and is now a couple of months old.

The next stage at which disorders of internal secretions are likely to be of importance, is puberty. The number of vague ailments amongst girls, especially at this stage, is very striking and analysis of the urine will invariably reveal chemical changes with bacilluria and probably pus. From puberty onwards girls are subject to a regular monthly cycle where vital changes are going on in their internal secretions and consequently in the chemical constitution of their blood, especially during the week preceding the menstrual onset. This condition certainly accounts for some cases of dysmenorrhœa and it is a most striking fact that exacerbations of pyelonephritis in women almost invariably take place in the pre-menstrual week.

The next stage at which derangements of internal secretions are most striking, is at the climacteric. Here you will find almost every condition of ill-health in women associated with chemical changes in the urine, generally of a very acid nature.

The third and last classification I have made, I have labelled fatigue, whether due to mental or physical agencies. Its appliciation is so general that I need hardly refer to it any further.

To sum up, my general conception of renal infection is somewhat this: At certain stages during life disturbances in metabolism are likely to take place, due to causes such as I have endeavoured to indicate. At such times the blood is filled with incomplete products of metabolism, generally acid in character and of a highly irritating nature. These are in due course eliminated by the kidneys with a varying degree of renal impairment. During life absorption of bacteria into the blood stream takes place from various foci, including the bowel, teeth, tonsils, pelvis, etc.; the bowel is the most common. These bacteria are likewise passed out of the circulation by the kidneys, also with a varying degree of damage to the kidney substance. It only wants these two conditions to coincide to a sufficient extent for renal infection to take place. And if there is obstruction to the urinary outflow in any way whatsoever, the presence of bacteria alone would be sufficient to cause infection. Obstruction to the urinary outflow is the most important feature in preventing recovery, although in all obstinate cases diligent search must be made for foci of infection as well.

You may think I have laboured unduly, but I feel sure that it is only by some such way of looking at these cases that a diminution may be achieved in their numbers. For there is no more pathetic figure in medicine than a miserable patient with pyelonephritis which resists all attempts at treatment.

REMARKS ON URINARY INFECTIONS.[1]

By S. F. McDonald, M.D., B.S. (Melb.), M.R.C.P. (London),
Honorary Physician, Hospital for Sick Children, Brisbane.

The whole question of pyelitis is one of the most difficult, important and depressing in medicine. It is a condition too often regarded lightly and too often quite unrecognized. What its prime mechanism may be, I think that we still do not understand. What its effects are, we are only slowly beginning to discover. It is some of these more remote effects which I wish particularly to emphasize.

Firstly, there is the question of so-called neuritis. "Neuritis" is a bad name—as bad as "gastritis"—and is too often a cloak for ignorance. We assure a man with a shooting pain in his arm that he has neuritis, without even pausing to consider whether the pain corresponds in any way to a nerve distribution. More often, if we look, we shall find the pain is localized to a muscle, just as it is, for instance, in the lumbar pain of typhoid or typhus. This is an exceedingly stubborn condition and in at least two cases I have seen it associated with pyelitis; I say advisedly "associated with," not "caused by." The muscle pains did clear up as the pyelitis cleared up, but I do not know (*i.*) whether it was the kidney condition that caused the muscle pain, (*ii.*) whether

[1] Read at a meeting of the Queensland Branch of the British Medical Association on September 1, 1922.

it was a general condition which affected both kidney and muscle, (*iii.*) whether it was a focal condition, *e.g.*, dental, which infected kidney and muscle and (*iv.*) whether anything I did had any real influence in curing the patient's condition.

Next we have the condition so exceedingly common in Queensland of œdema of the feet in hot weather. It is worth examining the urine of every one of these patients. Pyelitis is associated with it sometimes. The balancing of *post hoc* and *propter hoc* is always a valuable mental exercise.

It is hard indeed to decide when to stop in considering the remote effects of pyelitis, but let me implore you never to forget that so-called neurasthenia is very often caused by a chronic toxæmia.

In no other disease is it more important to remember Freud's advice, that a thorough search should always be made before any patient is stated to be suffering from a neurosis.

Dr. Roe will easily recall one patient in whom this phase was the most pronounced. A woman of twenty-seven, apparently always healthy, began to complain of headaches, vague pains, "nervous feelings," worry, jealousy, sleeplessness, nightmare and a general condition of misery which was wearing out herself and her family. She was sent to me as a "nervous patient" by a man in whose judgement I had much confidence. On the surface the picture was one of severe "anxiety hysteria" and the condition had lasted three years. I was called to see her one night for intense pain in the right side and a general feeling of wretchedness, palpitation and impending death. She had an enlarged, very tender and easily palpable right kidney. Her urine was loaded with pus cells. Her temperature was 39.4° C. (103° F.). With all my previous reservations, under treatment directed to the pyelitis, *viz.*, rest, alkalies and some local treatment carried out by Dr. Roe, she is now much better; there is, indeed, the "superadded functional," but that, too, the product of three years' wrong treatment and reaction, is diminishing.

It is worth your while to remember that very dreadful results may come from (*a*) treating a functional condition as organic, (*b*) treating an organic case as functional. I leave the explanation to my friend, Dr. Lowson.

With regard to treatment, I fear my attitude of philosophic doubt becomes more and more pronounced. I have discussed this matter frequently with Dr. Roe. I wish I shared his optimism and his confidence. It is true that many of these patients do well on a definite line of treatment, especially alkalies; others do not. My own theory, very tentatively put forward, is that we are dealing with different forms of organisms. I cannot accept the *Bacillus coli communis* as the whole source of our troubles. I cannot even believe that an alkaline urine is prejudicial to the *Bacillus coli communis*, which will grow under many conditions.

In regard to the condition spoken of by Dr. Roe as acidosis, I am quite ready to believe that many patients in whom the urine is acid, do well on an alkaline line of treatment. My own suggestion is that the causal organism in these cases cannot live in an alkaline medium.

The reaction of the blood is a very delicately controlled thing, as Haldane has pointed out. And it seems difficult to believe that the extraordinarily sensitive, yet sturdy, mechanism of respiration acid, alkali, carbon dioxide and hydrogen-ion concentration can be upset by one crude administration of alkali. If you will read Joslin, for instance, on the "Acidosis of Diabetes" you will see my point. That the administration of alkalies often does wonderful things everyone will admit. But the infecting organism may grow tolerant and may even be more affected by the alternate administration of acids.

One small girl of six originally yielded at once to alkalinization of the urine, then her parents began to administer alkalies on the slightest suspicion of pain, fever or malaise, but each attack progressed and ran a definite course quite unaffected. Now hexamine and acids are much more effective.

I have one brilliant success from the injection of collargol into the renal pelvis; I know of many failures. Of vaccines I know little, none of it successful. What I have said seems hopeless and discouraging. I have been watching a patient with pyelitis for the past four years; until three months ago she presented the severest manifestations in spite of the most devoted efforts by her advisers and seemed merging into a hopeless invalidism. Two months ago, after a month's very severe attack, everything changed; for no apparent reason the whole condition has altered and almost normal health has returned. The essential facts that we must remember are that the condition is a crippling one to patient and family alike, that much of it is unrecognized, that it is exceedingly stubborn to treat and that it is our duty to endeavour to alleviate if we cannot cure. Next to the hysteric, I believe that more of these unfortunates than any other class form the *clientèle* of the unqualified practitioner. Finally, when we do achieve a cure, let us ask ourselves whether we should not more than ever quote Ambroise Paré: "I dressed the wound; God cured it!"

URINARY INFECTION IN CHILDREN.[1]

By D. Gifford Croll, M.B. (Sydney),
Brisbane.

Urinary infection in infants and young children, usually due to the *Bacilli coli* organisms, is much more common than is generally supposed. It is usually missed because it is not considered in making a diagnosis. Once considered, the diagnosis is easily settled, if a microscope is available. No skilled pathologist is necessary and no technique is required. All that is needed is to place a drop of urine on a slide and to look at it with a low power objective, do not look for bacteria, look for pus cells! For a man not versed in pathology there

[1] Read at a meeting of the Queensland Branch of the British Medical Association on September 1, 1922.

would be several sources of error in making a diagnosis by bacteria. The urine might not be perfectly fresh and contain foreign organisms; oscillating inorganic bodies might be mistaken for bacteria; typhoid or other bacilli might be present. In practically all cases of primary urinary infections pus cells are present and the only thing likely to be confused with them are red blood corpuscles and epithelial cells from the neck of the bladder, both of which are comparatively rare. Pus cells are easily distinguished at once, even amongst a lot of débris, and they remain distinguishable, even when the urine is a day or two old.

A diagnosis cannot be made without a microscope. A cloudy urine may be due to secondary bacterial infection, inorganic deposit, mucous or epithelial cells. An apparently clear urine may be shown under the microscope to contain pus cells.

Now what are the symptoms that would lead you to make a microscopical examination of the urine? Text-books generally describe the acute conditions, sudden onset with high fever and profound constitutional disturbance and possibly cerebral symptoms. Such cases undoubtedly occur, but the type of case commonly met with in practice is entirely different. Possibly it originates in an attack such as described, but the fact has been forgotten or overlooked by the mother.

The commonest symptoms are pallor, malnutrition, occasional fever and sleeplessness. I have frequently seen these patients treated for dyspepsia, carefully and patiently dieted for months without any improvement and then a cure is promptly effected by a few doses of potassium citrate and "Urotropine." Even if only one or two pus cells are discovered, they cannot be ignored. A baby eighteen months old was brought to me on account of sleeplessness; I had previously treated it for pyelitis. It looked a healthy baby and examination of the urine revealed less than one pus cell to a field (two-thirds of an inch objective). I could find no other cause for sleeplessness, so I placed it on 0.6 gramme (ten grains) doses of potassium citrate and 0.06 gramme (one grain) doses of "Urotropine" every four hours. The effect was magical. The sleeplessness disappeared in two or three days and yesterday the mother informed me that it returned once or twice, but was easily remedied by another bottle of the medicine.

Unlike the urinary infections of adults the treatment is easy and generally effective. I usually give six decigrammes of potassium citrate every two hours at first, then every four hours and finally three times a day when the urine has become alkaline. If this is not effective, I add "Urotropine" or "Hexamine" to the potassium citrate mixture. I do not know which is the better, "Urotropine" or "Hexamine"; I find them both effective, even in an acid urine.

If pus persists in spite of the above treatment, I should suspect the presence of a calculus or possibly of tuberculous kidney. Twice I have seen calculus in a child under two years of age. I do not think I have seen a tuberculous kidney at that age.

THE ÆTIOLOGY OF URINARY INFECTIONS.[1]

By J. V. Duhig, M.B. (Sydney),
Honorary Pathologist, Mater Misericordiæ Hospital, Brisbane.

Infections of the urinary tract, ætiologically considered, fall into two groups: (*a*) infections with obstruction, (*b*) infections without obstruction.

Group (*a*) is the more numerous, comprising cases due to obstruction directly or indirectly. With this classification in view, it is easy to see how all grades of infection with resultant tissue destruction may occur from an extensive pyonephrosis through pyelonephritis and a simple non-suppurative pyelitis to a transient bacilluria.

Infection with Obstruction.

It is immaterial to this paper to enumerate or discuss the forms of obstruction to the urinary flow which may occur, though it is important not to overlook those which are not immediately apparent, such as the pregnant uterus, a bowel distended by constipation and other forms of abdominal tumour altogether remote from the urinary tract. I am here concerned mainly with the results of these obstructions.

In its simplest and most obvious result obstruction leads to hydronephrosis, uni- or bi-lateral, and of varying degree of severity, depending upon the completeness and site of obstruction. With the advent of virulent bacteria the contents of the hydronephrotic sac become purulent—pyonephrosis. A similar condition occurs when there is infection about a renal calculus. These conditions occur in complete or nearly complete obstruction. When the obstruction is incomplete, lesions of a less severe nature are encountered and the inflammatory reaction predominates over the mechanically produced distension associated with complete obstruction and hydronephrosis. These lesions are pyelonephritis and pyelitis. The conditions are not strictly separate entities, but stages of the same process, due most commonly to obstruction and stagnation of urine, so frequently seen in women who are constipated or pregnant or both.

In these conditions there are abscesses in the kidney itself, with an intense inflammation of the mucosa of the pelvis. The abscesses are usually in the cortex and are small, as a rule, though they may conglomerate and show a distinct bulge on the surface, which, when the capsule is stripped, exudes pus. There is usually much destruction of tissue to the point of obliterating the normal appearance. Sometimes the extension of the abscess down the ureter is noted.

Pyelitis may, of course, occur independently of any change in the kidney substance and here there are again gradations from inflammation of the pelvic mucous membrane to suppuration and abscess formation.

Infections without Obstruction.

Now all these conditions, except, of course, hydronephrosis, may occur without any obstruction or

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any disease in the lower urinary tract at all, *e.g.*, infections without obstruction, and discussion has long raged about the point as to how the bacteria get to the focus.

Spread of Infection.

Let us again divide the conditions, now according to site.

A pyelitis may occur concomitantly with, not necessarily secondary to, a cystitis or independently, as I have said of any lesion in the lower tract. If there are concomitant cystitis and pyelitis, how do the organisms get to the pelvis or bladder. If, and it is not agreed this is so, the bacteria first lodge in the bladder and settle down later in the kidney, what route do they take? We have a choice of theories, none of them quite satisfactory. Some believe the organisms enter the blood stream from the bladder and are deposited in the kidney. Others regard the route as along the peri-ureteral lymphatics and veins. It would be difficult to conceive of a venous spread, as that view would involve retrograde embolism—a very far-fetched idea and one not lightly to be called in aid. Another view is that bacteria pass up the ureter against a constant flushing stream of urine, which is possible if there is a stagnant column of urine above the obstruction. But if there is no obtruction, how do we stand? My own view is this, that the bacteria are blood-borne, not from the bladder necessarily, indeed hardly at all, as I frequently see cases of cystitis in old men with large prostates and much residual urine with perfectly healthy kidneys. Calmette and others have shown that migration of bacteria from the intestinal tract is at a minimum when the bowel is at rest and the mucosa uninjured, but that after a heavy meal and under the influence of excessive peristalsis, this migration reaches a very high degree and becomes excessive in the case of trauma of the bowel. The presence of intestinal flora may be demonstrated in the blood serum. This being so, curative horse serum is never withdrawn, except after the animal has undergone a long period of testing to increase the chance of securing a perfectly sterile serum.

The fact of the predominant organism (in uncomplicated primary pyelitis or pyelonephritis it is the *Bacillus coli* and in the frequency of pyelitis in children or even adults after an attack of enteritis of whatever kind or consequent upon constipation) leads me to believe the organism is blood-borne to the kidney, its natural outlet once it has entered the blood stream. The view is feasible and simple and the course of events is actually that seen in pyelitis or pyelonephritis secondary to an acute infection, either as part of a pyæmia or from a focus elsewhere, as in the heart. In this condition abscesses develope about bacteria brought to the kidney by the blood in an effort to excrete them. This is true of streptococcal or staphylococcal nephritis and may be true of pyelitis due to *Bacillus coli* of a simple and uncomplicated kind.

It is harder to explain the path of infection in pyelitis existing at the same time as, but of a later development than, cystitis where there is obstruction and still harder to explain pyelonephritis secondary to pyelitis. I am inclined to believe the infection is partly an ascending one, partly hæmatogenous in the case of pyelitis. Having explained the pyelitis, how do we account for the infection of the kidney? There are three routes, the tubules, the arteries and the lymphatics. Direct infection of the tubules seems the most feasible and has experimental data to back it, Schmidt and Aschoff having obstructed the ureters after artificially infecting the pelvis and later finding the tubules plugged with bacteria.

The organism most frequently incriminated in kidney infection is the *Bacillus coli*, others less frequently found being streptococcus and staphylococcus, pneumococcus, etc.. Of course, any organism may occur in the urine in the course of any infective process, as *Bacillus typhosus*, *Bacillus dysenteriæ*, etc..

Cystitis is usually the result of the catheter, which is the starting point of a vicious circle. For some reason normal emptying of the bladder is impossible or incomplete. The catheter carries in bacteria to the urine, which normally is sterile and an ideal culture medium, kept constantly at blood heat by the human incubator. The urine becomes turbid, alkaline, full of débris and cystitis is set up. I have said the urine becomes alkaline, but not necessarily, and if it does, it is of no actual pathological significance, since the alkalinity is caused by non-pathogenic saprophytes.

The vicious circle is further enlarged by the occurrence of a dilatation, greater or less according to the nature of the obstruction. The walls stretch, as in old men, into diverticula. Along with this hypertrophy occurs from repeated efforts of the bladder to empty itself. If distension predominates over hypertrophy, then the residual urine becomes greater. Further, the entrance of the ureters is oblique through the muscle and distension tends to flatten out the ureters; it is therefore obvious that any obstruction to the outflow of urine from the bladder makes the inflow from the ureter equally difficult. The catheter should be taken up with the greatest care and trepidation; it is a two-edged sword and a lethal weapon in the hands of the careless and incompetent.

Bacilluria simply means bacilli passed in the urine with or without pus. Strictly I apply the term to the condition where there is no pus present. It occurs in the course of acute infections and in chronic cystitis. It may be transient or go on to cystitis and pyelitis.

Organisms Implicated and Sex Incidence.

For the eight months ended August 31, 1922, the samples of urine examined at the Mater Misericordiæ Hospital of patients suspected of kidney infection were one hundred and twenty-eight according to the following table:

	Number.
Sex—	
Male	32
Female	96
Organisms—	
Bacillus coli alone	80
Bacillus coli with others	17
Staphylococci	15
Streptococci	4
Others	12
Presence of Pus	105

There were fifteen cases of cystitis and three cases of bacilluria with no pus present. In this series 75% of the patients were females.

The *Bacillus coli communis* either alone or with others was present in 76% of cases. Most of the staphylococcal cases were cystitis, one being probably a catheter infection.

Diagnosis.

Freshly voided urine in cases of pyelitis is usually a little turbid, but sediments quickly when pus is present, leaving an almost clear supernatant fluid. As a rule the filtrate is clear, unless the number of organisms is excessive. Urine from cystitis is always turbid and sediments on standing; it is almost always alkaline in reaction and on filtration it does not become clear, but still retains its turbidity.

In a pure bacilluria no amount of filtering will render the urine clear. It is uniformly turbid and little, if any, deposit occurs on standing. After a time, of course, it becomes alkaline, with a heavy phosphate deposit.

The diagnosis is clinched by microscopical examination, which will reveal the presence of pus when the chemical test alone is negative.

Pus and bacteria in the urine are not diagnostic of simple primary urinary infection. The laboratory findings are ancillary to the clinical aspect and a diagnosis of pyelitis and pyelonephritis should be made on the presence of pus and bacteria in the urine when other possibilities are excluded, as typhoid, fever, dysentery, pneumonia, etc..

THE ORGANIZATION AND WORKING OF A GENITO-URINARY CLINIC IN CONNEXION WITH A TEACHING HOSPITAL.

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The following is a description of the organization and working of the University of California Genito-Urinary Clinic and of the facilities offered there for urological instruction.

The material available for purposes of instruction is drawn from the following sources: the dispensary service of the University of California Medical School and the surgical services of the University of California Hospital and the City and County Hospital. In addition, the urological services of the Hahnemann Hospital, St. Luke's Hospital and the Children's Hospital come under the administration of the department, but are not used as yet for teaching purposes.

The University of California Medical School Out-Patient Department.

During last year 10,000 out of a total of 88,000 patients were treated in the urologic division of the Out-Patient Department. This service forms the chief source of supply for the surgical beds in the hospitals.

It is divided into two separate clinics, one for men and one for women, which are run on the same lines as the medical, surgical, gynæcological and other clinics.

All persons, providing they satisfy the Social Service Department that they are *bona fide* hospital patients and not able to pay for treatment, are admissible as patients.

It is open every morning, excepting Sundays and public holidays, from nine to eleven o'clock. In addition, there is a night clinic on Monday, Wednesday and Friday evenings, from seven to nine o'clock.

The Out-Patient Clinic is in charge of locally practising urologists, who act as instructors. These men, of whom there are four, are in charge on alternate days, two each for the men's and women's clinics respectively. Some of these have additional private appointments as visiting urologists to the Hahnemann, St. Luke's and Children's Hospitals.

The service at the University of California Hospital consists of two hundred and sixty-five beds, eighty of which are in private service, the remainder being ward beds. The Department of Urology has four clinical beds, otherwise the entire service is composed of the chief's private patients.

The City and County Hospital has a total of approximately one thousand beds. Of this number, twenty are allotted to the urologic service. The supply of surgical "material" for these beds is obtained chiefly from the University of California Medical School Out-Patient Department, together with referred patients from other sections of the hospital. The staff is composed of the chief of the department and six instructors, all of whom are practising urologists in the city of San Francisco. Men in training are appointed as assistants to the regular staff and this appointment holds for the three or four years' period of training. These men, in addition, receive appointments relative to the respective positions of their rotation service, such as Resident Urologist of the University Hospital, Visiting Urologist of the City and County Hospitals. Assistants alone get a salary, four of which at $600 a year are available.

Training in the Department of Urology.

The men receiving training in the Department of Urology come under three headings: (*i.*) students, (*ii.*) specialists and (*iii.*) post-graduates.

(i.) Students.

All students of medicine receive a course in urology, consisting of a series of lectures on the fundamentals of the subject, without much detail, but containing such matter as the general practitioner should know. This series of lectures is given during the second semester of their third year (corresponding to the second term of the fourth year at Sydney). During the first semester of their fourth year (corresponding to the first term of the fifth year at Sydney) all students are required to put in three weeks in the Genito-Urinary Out-Patient Clinic.

Attendance is essential for two reasons: (*i.*) there is an examination in urology at the end of the year, which they are obliged to pass; (*ii.*) if they fail to put in the required time in the clinic, they are reported by the man in charge for non-attendance and,

unless this absence is satisfactorily explained, their failure in urology will result.

During their term in the Out-Patient Clinic they receive instruction in urology from a clinical standpoint. Lectures are also given on any interesting current material which may be on hand.

Students, under the supervision of the men in charge, treat specified cases, commencing with the history taking and then following the case up. They are encouraged to form a diagnosis; their reasons for coming to such a conclusion are asked; they are required to state what treatment they suggest and, when practicable, they are permitted to carry out such treatment.

Students are not permitted the use of the cystoscope. Cystoscopy is performed entirely by the men in charge, who may demonstrate various typical and atypical conditions to the students.

During their fourth year all students are given the option of taking a six weeks' course in any special subject they desire. If urology is selected, the student has an additional term of six weeks in the Out-Patient Clinic, taking over cases, working them up and carrying on routine treatment.

Students are at all times admitted to the operating theatres, where they may witness any operations that are being carried out; but they do not assist at such operations, except in the capacity of an interne, while on the Urological Service.

(ii.) Specialists.

Under this heading come those men who intend specializing in urology. A man before entering on this course must be fully qualified and have had a year's internship. Such a man is taken only on the understanding that he will serve for a term of at least three years, during which time the work is subdivided up as follows:

During the first year he serves as an assistant in the Out-Patient Clinic every morning, doing routine work, but not being permitted the carrying out of cystoscopic work other than observation and diagnosis.

After six months in this clinic he is given a free hand, with the right to treat patients and conditions as he thinks fit.

Three afternoons in each week he gets private "office" work, where he assists and is given an insight into the workings of a successful urological practice.

During the second year he is appointed Resident Urologist of the University Hospital, where he has an interne under him and has complete charge of all operative and post-operative cases. During this time he acts as assistant in all operations, supervising and being responsible for the after-treatment.

In the third year he serves first as junior and then later has complete charge of the urologic service at the City and County Hospital. While on this service he holds a similar position at the University of California Medical School Out-Patient Clinic, from which source the greater number of patients admitted to the surgical wards of the hospital is drawn.

In this way the "specialist" sees the patients first in the Out-Patient Clinic and if surgical treatment is indicated they are referred to the City and County Hospital.

During the first six months of this appointment the time is devoted chiefly to pre-operative, operative and post-operative work, all ground work being done by the interne on the service. Minor operations and cystoscopic examinations are performed, together with assisting at the major operations.

During the last six months the "specialist" is in complete charge of the entire urological service, as well as being consulting urologist to the rest of the hospital, all operations and work considered necessary being performed.

Research work forms a very important part of the urological training and is at all times greatly encouraged. The department is very fortunate in this respect in having the Hooper Research Building attached to the University of California Hospital, one section of which has been given over to the urological service.

During each of the three years of urological training a definite time is allotted to research. The subject may be some suggestion from the chief or some original idea of his own. In either case all materials necessary are supplied by the department and every encouragement given.

(iii.) Post-Graduates.

By post-graduate is meant the general practitioner who is desirous of entering the urological service for a term, such as three or six months, with the intention of gleaning the elements of urology and learning the technique of cystoscopic examinations.

There is no provision made for this class of man. Such a teaching is regarded as detrimental to the interests of urology and would be injurious to the reputation of the school where such a smattering was given.

Staff Meetings.

The staff is at all times kept fully posted in everything that is done in the department. This is brought about in several ways. For instance, there are weekly ward rounds at the City and County Hospital, when all cases are gone over by the chief and all members of the staff. Examinations are carried out and suggestions offered. This serves to keep even those members of the staff who are not at this hospital, acquainted with everything of interest being done there and is a check on the work being done by the junior and senior men in charge.

Every month there is a urological seminar, which is open to all members of the hospital staff. At these meetings short lectures are given or some work is presented by a member of the staff. This work usually comprises some special subject on which the lecturer has been doing research work. All articles are presented at these meetings before general presentation at some regular medical meeting and before publication.

Weekly clinical-pathological conferences are held, attendance at which is not open to the entire hospital staff, but is confined to post-graduate members of the Urological Department. At these meetings the correlation of the current clinical material of

the University and City and County Hospitals with pathological findings is discussed, together with any outstanding current literature of interest.

The Department run as stated is capable of managing the training of six men simultaneously, two of whom are in their first, two in their second and two in their final year's training. This organization, which is the outcome of the sincere and untiring energy of its chief, has been gradually evolved and now forms a harmoniously working Urological Department which is efficient and satisfactory in every detail.

Requirements for Training in Urology.

The following is the outline drawn up by the Committee of Urologists recently appointed by the American Medical Association for the training of urologists. It will be seen how closely the above organization conforms to it.

Urology should be understood to embrace diseases of the urinary organs in men, women and children, as well as the genital and sexual diseases of the male. The various surgical lesions of the above-mentioned organs are to be included.

The minimum requirements for training in urology are:

1. Graduation from a first-class medical school.

2. Internship of two years' duration, which may be divided in one of two ways:

(*i.*) (*a*) A medical internship for one year. This to be a broad general service with men of wide experience. (*b*) A surgical internship for one year in a general surgical service, with ample opportunity to perform the more common standard operations. This under supervision and instruction of the attending surgeon.

(*ii.*) (*a*) Medical internship for six months. (*b*) Surgical internship for six months. (*c*) Gynæcological internship for six months. (*d*) Surgical resident for six months.

These services to be taken in a standard hospital with large clinical material and proper supervision.

3. Special urological training. This to embrace two years or preferably three years of special training in urology in a first-class hospital. It should be offered only at those institutions that have fully equipped departments of urology with large and varied material, including men, women and children, with ample opportunity to perform major urological operations.

(*i.*) Work in the out-patient department.

(*ii.*) Laboratory work, to include bacteriology and surgical pathology of the genito-urinary system, blood chemistry, etc..

(*iii.*) Extensive experience in the use of special diagnostic and therapeutic instruments, such as the cystoscope, the ureteral catheter, the urethroscope, the fulguration apparatus, pyelographic work, functional tests, etc..

(*iv.*) Röntgenography. (*a*) Primarily of the genito-urinary tract; that is, combined urological and radioscopical studies, by means of pyelography, cystography, etc.. (*b*) Secondarily, some instruction in Röntgenography as related to certain conditions of the genito-urinary tract, such as bone metastasis in malignant diseases, bone changes in arthritis, etc..

(*v.*) Ample facilities for research and experimental urology.

(*vi.*) Pre-operative and post-operative care of patients with uro-genital disease.

(*vii.*) During the period of training, at least six months should be spent as a resident urologist, with extensive opportunity to perform major urological surgery or to act as an assistant to one experienced in urological surgery.

OBSERVATIONS UPON THE POSSIBILITY OF SCHISTOSOMIASIS BECOMING ESTABLISHED IN AUSTRALIA.

By Frank Milton, M.R.C.S., L.R.C.P.,
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Human schistosomiasis is widely distributed in both the old and the new worlds, but is limited to a belt lying between 38° north and 36° south of the Equator and Australia, with the exception of a large slice of Victoria and a small portion of New South Wales, lies wholly within this belt.

Both *Schistosomiasis hæmatobii* and *Schistosomiasis mansoni* are endemic in Egypt and many of the Australian troops, whilst stationed in that country during the late war, contracted the disease and were repatriated while still passing living eggs of the parasites in their excreta.

I have, unfortunately, no record of the number of men who were known to have been infected, but Fairley[1] in 1919 recorded seventy-five infected troopers of the Australian Light Horse who came under his care in 1916 and as these all belonged to one unit only and were all infected within a short space of time, the total infestation of the whole Australian Force, spread over the whole time spent in Egypt, was probably very large indeed.

Schistosomiasis in the vast majority of cases gives rise to symptoms of so mild a character that those suffering from the disease are unconscious of their infection. It is unlikely that these excessively mild cases should be the rule in a race newly infested and with no acquired racial resistance, but it is quite fair to presume that a number of such mild infections did occur amongst the Australian troops and may have passed unidentified and unrecorded, although their potentiality for the infection of others is as great as that of the more severe cases.

Further, many cases are on record where the disease appears not to have revealed itself until long after the last possible moment of infection. This period of latency appears to have extended to as much as three or even four years. Given this latent period, it is quite possible that cases may have occurred amongst the troops who only developed symptoms after demobilization when they could be no longer traced by the authorities and who may now be evacuating fertile eggs in many unsuspected localities.

Most of the returned troops were demobilized before the curative treatment by tartarated antimony

was in general use and the infected and infective men were returned to their homes still passing living schistosome eggs in their evacuations.

It is known that efforts were made by the Defence Department to recall all men for radical treatment as soon as the possibilities of this were recognized, but with what degree of success these efforts were rewarded is not known.

Sporadic indigenous cases are known to have occurred within the Commonwealth after the South African War, when the number of returned troops was much smaller than those returning after the late war.

The length of life of the mature parasite is not accurately known, but it is established that they can survive and go on producing fertile eggs for a period of from twenty to twenty-five years and probably more.

In the face of all this the probability is that there are at this moment a considerable number of infected persons distributing fertile eggs over many parts of the country.

The human schistosomes are digenetic trematodes needing for their development certain fresh water molluscs as their intermediate and man as their definitive hosts; they are of three distinct species, *Schistosoma hæmatobium, Schistosoma mansoni* and *Schistosoma japonicum.*

For the sake of simplicity we may neglect the far eastern schistosome, *Schistosoma japonicum,* for, although this may have been introduced into the Northern Territory by Asiatics, it is not concerned in the question of the possibility of the infection of the country by returned troops.

Leiper, in Egypt,[2] in 1915 showed that *Schistosoma hæmatobium* developed in fresh water molluscs of the genus *Bullinus* and that *Schistosoma mansoni* developed in its near relative, *Planorbis.*

Bullinus and *Planorbis* both belong to the order *Pulmonata* and the family *Planorbidæ.*

It was shown at the same time that not all species of these genera carried or appeared to be able to carry the parasites.

The species of *Bullinus* found to harbour *Schistosoma hæmatobium* were identified as *Bullinus contortus, Bullinus dybowski, Bullinus innesi* and *Bullinus alexandrina.* Malacological nomenclature is not a thing to be lightly touched by the amateur, but it would appear that *Bullinus dybowski* and *Bullinus innesi* are merely local races of *Bullinus contortus* and that *Bullinus alexandrina* is synonymous with *Bullinus dybowski*; if this is so, it means that *Bullinus contortus* is the sole host of *Schistosoma hæmatobium* in Egypt.

Schistosoma mansoni is carried in Egypt by *Planorbis boissyi.*

The questions now arise:

Do *Bullinus contortus* and/or *Planorbis boissyi* exist in those parts of Australia which are suited to the existence and propagation of schistosomes?

If not, can *Schistosoma cercariæ* adapt itself to other species or genera of molluscs indigenous in Australia?

Both *Planorbis* and *Bullinus* are cosmopolitan and very widely distributed genera, though there are queer gaps even in their distribution and one of them, *Bullinus* and its synonyms *Isidora* and *Isidorella,* together with its near relative, *Physastra,* certainly exist in Australia. Of *Planorbis* I am ashamed to have to plead ignorance, but I take it that it does occur, though it is unlikely that the species of either *Bullinus* or *Planorbis,* which are the hosts of the parasites in Egypt, should exist in Australia; the answer to our first question is probably in the negative.

At first it was held that the molluscs infected were specific for their particular parasites and that any country free from these species of snails, would be free also from its specific parasite.

In 1917, however, Iturbe and Gonzales[3] found that, though *Planorbis boissyi* did not occur in Venezuela, *Schistosoma mansoni* was very common in the country and was carried by another *Planorbis, Planorbis guadelupensis,* whilst in 1919 Lutz[4] found two other species of *Planorbis* infected in Brazil, *Planorbis olivaceus* in Sergipe and Bahia and *Planorbis centimetralis* in Pernambuco and Parahyba, whilst the host in Rio Grande do Norte and Ceara was still *Planorbis guadelupensis.*

In 1920 Annie Porter[5] found that in South Africa *Schistosoma mansoni* was carried by yet another species, *Planorbis pfeifferi.*

In 1921 Nicolle and Gobert[6] sought to incriminate *Bullinus brochii* with carrying *Schistosoma hæmatobium* in Southern Tunisia; the circumstantial evidence was very strong, though no specimens were actually found infected with the parasite.

Up to this point we have *Schistosoma mansoni* adapting itself to five different species of *Planorbis* and *Schistosoma hæmatobium* probably to two species of *Bullinus.*

In 1919 Annie Porter[7] found both *Schistosoma hæmatobium* and *Schistosoma mansoni* parasitic in quite a new snail, *Physopsis africana.*

Physopsis is, probably, a sub-family of *Bullinus,* although its true position has not been clearly worked out. This for *Schistosoma hæmatobium,* although a striking was still not a very wide departure from its natural habits, but for *Schistosoma mansoni* it was a new venture altogether.

In 1921 Bettencourt[8] and his colleagues,, working in Portugal, found that *Schistosoma hæmatobium* had rivalled or even outdone this change over on the part of *Schistosoma mansoni,* for they found that its host in that country was a true *Planorbis. Planorbis corneus,* variety *metidgensis,* the generic host of its rival, *Schistosoma mansoni.*

In 1919, however, Annie Porter[7] had discovered the most striking of all these adaptations, for she found that in South Africa *Schistosoma hæmatobium* had adopted as its common host *Limnæa natalensis,* a pulmonate mollusc, but of a different family (*Limnæidæ*) to both *Planorbis* and *Bullinus* and this although *Bullinus* (*Isidora*) is common in the country.

We have thus a very wide range of adaptability exhibited by the two schistosomes which are known to have been introduced into Australia, and it is quite admissible to believe that these parasites will be found to be able to adapt themselves further to

some of the species of the *Planorbinæ*, *Bullininæ* or *Limnæidæ* which they will find in the Commonwealth and it would seem that the danger of the establishment of schistosomiasis as an endemic disease in Australia is imminent.

Is There an at Present Unrecognized Influence Regulating the Geographical Distribution of Human Parasites?

Other things besides suitable hosts, climate and conditions of life would appear to be necessary for a parasite to obtain a footing in a new country.

It would seem that there is some as yet unidentified power limiting and prescribing the geographical distribution of human endo-parasites, as is suggested by the limitations of the tapeworms, the hookworms, micro-filariæ, trypanosomes, *et cetera*.

It would seem also as if, when once a particular species of a parasite has gained a footing in a country or district, it becomes impossible or else extremely difficult for any other species of the same parasite to establish itself therein. This is particularly observable in the cases of the schistosomes and the hookworms.

Schistosoma japonicum is strictly limited as an endemic disease to China, Japan and the Philippine Islands. Fertile eggs of this species have been carried to many other regions of the world, yet nowhere else has any trace of the disease been found, nor can any other schistosome establish itself in the Philippines, Japan or China.

Schistosoma mansoni is found occupying South America, the West Indian Islands and the basin of the Congo to the exclusion of both the other species.

Schistosoma hæmatobium is the only form found in Western Asia, the Hedjaz, Mesopotamia and Persia and was until quite recently the only species found in South Africa. It predominates in all the coastal regions of Africa, but is here accompanied by a small number of *Schistosoma mansoni*, though nowhere are the two species found together in anything approaching equal numbers.

On the other hand—and this is particularly interesting to us—there are vast regions within the bilharzia belt where no species of the parasite has ever succeeded in establishing itself, the largest and most noteworthy of these being India, in its widest geographical sense, the Southern States of the American Union, together with Mexico and the States of Central America.

Schistosomiasis is known by actual demonstration of its ova in the kidneys of mummies[9] to have existed in Egypt as early as 1250 B.C. and before this there was an established trade between Egypt and India *viâ* Arabia and Persia as well as directly by sea.

It is probable that throughout more than three thousand years infected persons have been constantly passing from Africa to India, carrying with them and distributing around them the eggs of the parasite; pilgrims from Mecca and Medina and the holy places of Mesopotamia are constantly bringing back the disease with them and it is known that schistosomiasis has been frequently introduced by infected troops, both after the South African and during the great war, and yet the disease has never taken root in the country and the sporadic cases which have been reported can almost be counted on the fingers of one's two hands.

Resistance of North America to Infestation.

The West Coast of Africa is intensely infected, principally with *Schistosoma hæmatobium*, throughout its whole extent and for a couple of centuries or more slaves from this infested coast were being poured into the West Indian Islands and the Southern States of North America, yet in the West Indies only one species of the parasite (*Schistosoma mansoni*) is found and in the United States no trace of either species appears to exist, either amongst the white people or amongst the descendants of the slaves themselves.

It has always been taken for granted that *Schistosoma mansoni* has been introduced into the new world from Africa, but I am inclined to think that things have worked the other way about and that *Schistosoma mansoni* was the indigenous American form of the parasite and that it passed from America to Africa and has since been fighting an uphill battle with *Schistosoma hæmatobium*, the true African form, that it has failed to gain a firm hold on any part of that continent with the exception of the basin of the Congo and though a few cases have struggled as far as the east coast, where it is extremely rare, it has never been able to cross from Africa to Asia.

If *Schistosoma mansoni* be indeed the aboriginal American species, this may supply the reason why *Schistosoma hæmatobium* has never gained a footing in the new world, but it does not explain why *Schistosoma mansoni* itself is absent from the Southern States and from Central America.

We have it on the authority of Asa C. Chandler[10] that "many species of *Planorbis* and *Limnæa* which could probably act as intermediary hosts, as well as the species in which the development has already been observed, are abundant in the United States" (I am a little doubtful of the accuracy of part of the statement), so that it is not for want of suitable intermediary hosts that the parasite has failed to establish itself.

The case of the West Indian Islands is, in a way, even more striking. All the larger islands of the Lesser Antilles and many of the smaller from Barbadoes to Antigua and then on to St. Kitts, St. Martin, the Virgin Islands and Puerto Rico are infested, many of them heavily, but from here on the infestation suddenly stops and neither San Domingo, Cuba or the Bahamas are able to show a single case among them.

There appears to be no reason for this; it is true that for some equally obscure reason *Planorbis guadelupensis*, the carrier of *Schistosoma mansoni* in the islands, is said not be found in Cuba, but it is replaced by the closely allied forms, *Planorbis caribeus* and *Planorbis affinis* and there should be little difficulty in *Schistosoma mansoni* adapting itself to one of these.

The Case in India.

Ever since it was realized that India was in danger of being infected with schistosomiasis by returning soldiers, a search has been carried on for cases of the disease and for possible molluscan hosts.

Up to now no indigenous centres of infection have been found and the search for molluscan hosts has revealed the fact that *Bullinus* is entirely absent from the country[11] and that the only species of *Planorbis* present is *Planorbis exustus* and one of the leading malacologists of India refuses[12] to admit this form to be a *Planorbis* at all and has created a new genus, *Indoplanorbis*, for its accommodation, though possibly wrongly.

Search in past records has revealed twelve cases, all of *Schistosoma hæmatobium*, which have, in ten certainly and in the other two probably, originated in the country.

Of these twelve infections four are useless for our purpose, since their place of origin is not stated, but they are of interest since three out of the four occurred in 1903, 1904 and 1906, years in which parts of India were undoubtedly infected by troops returned from South Africa.

Of the remaining eight cases, two were in British soldiers who developed symptoms of bilharziosis in Allahabad in 1905;[13] three other British soldiers were infected in Mian Mir, Punjaub, in 1903,[14] 1904 and 1906 respectively; two British soldiers contracted the disease in Poona, Bombay Presidency, in 1906[15] and the last case was also in a British soldier, who picked up the disease in Secundrabad, Deccan, as recently as September, 1920.[16]

Of the other four cases, two were in British soldiers, both of whom had served in South Africa, leaving that country, the one nineteen months and the other three years and four months before symptoms developed in India. One was a native of Bombay who had never been out of that Presidency; he developed symptoms in 1903. The fourth was a Parsee girl living at Rajkot, Bombay Presidency, who was found to have schistosome eggs in her urine in 1914.

Of the two patients at Allahabad, both had been in South Africa, one having left that country twenty-eight months and the other thirty-two months before becoming ill.

Of the other patients—three at Mian Mir and two at Poona and the recent one at Secundrabad—none had been out of England before coming direct to India.

The two Allahabad patients might possibly have been infected during their service in South Africa, but, if they had, they had both carried the parasite well over two years without any symptoms, it being definitely stated in their medical history that they had both enjoyed good health and had never had any signs of urinary trouble before the symptoms appeared in Allahabad.

In the remaining six patients there is no doubt but that they were infected in India and at the stations named.

It results then that from 1903 to 1906 the molluscs at Mian Mir were infected and were capable of transmitting the disease, that during 1906 the molluscs at Poona were similarly infected and it is probable that during 1905 the molluscs at Allahabad were in the same condition.

No further cases have ever been reported from any of these stations since 1906 and it must be presumed that the infection among the molluscs died out naturally, since there is no record of any measures having been taken to free the stations from infection. It was not known at that date in what way schistosomiasis was passed from patient to patient.

During 1918-1919 a large number of Imperial Service troops invalided from Egypt with schistosomiasis were either under treated in the Hyslop War Hospital at Bolarum, near Secundrabad, Deccan, or living in the bazaar there.

In August, 1921, a British gunner entered the Station Hospital, Secundrabad, suffering from diarrhœa. An examination of his urine revealed the presence of terminal-spined schistosome ova. This man, who had never been out of England before leaving for India in March, 1920, came straight to Bombay without leaving the ship on the voyage, was entrained directly for Secundrabad and remained there until he fell ill in August, 1921. It was ascertained that in August and September, 1920, he formed the habit of going for solitary walks and of bathing in one of two small tanks near Bolarum; he had noticed that on each occasion he had suffered from itching all over his body after leaving the water.

The possibilities of this case were immediately recognized. Energetic search for other cases was made amongst the natives living and working in the neighbourhood of the incriminated pools and thousands of snails from these and other tanks in the vicinity were examined, but all without revealing any trace of infection in either man or mollusc.

Search is indeed still proceeding, but I think it may be taken for granted that the infection has died out naturally, in the same way as it appears to have done at Mian Mir and Poona.

We have then three or possibly four distinct occasions in which schistosomiasis has obtained a footing in India. In one of them it appears to have been able to persist for three years or more and yet in each case the infection has died out naturally, being for some unknown reason unable to continue.

Resistance to the Establishment of Schistosoma Mansoni in South Africa.

Though Bilharz in 1852 noted the existence of lateral-spined schistosome eggs, it was not until fifty years later that any special importance was attached to them. In 1902 Manson suggested their belonging to a species other than *Schistisoma hæmatobium* and in 1907 Samboni named this new species *Schistosoma mansoni*.

A controversy as to the validity of this new species raged from that year until 1912, the protagonists being Looss and Leiper; it rumbled on until 1916, when Looss, having silently faded away as the result of happenings in August, 1914, Leiper produced the final conclusive evidence of the specific difference between the worm laying the lateral-spined eggs and the one laying the terminal-spined eggs.

Up to 1910 or 1912 most observers grouped all cases of schistosomiasis together under the name of bilharziosis and it is not possible up to this date to affirm or deny the existence of either species in any particular place. But as far as South Africa is concerned, all cases described were clearly vesical, *i.e.*, terminal-spined, and the intestinal form was not recognized, except among imported labourers working in the Transvaal mines.

In 1908[17] Turner stated definitely that he had not found lateral-spined eggs in natives coming from other than tropical or semi-tropical parts of Africa and in 1921[18] Cawston said that: "Up to June that year no case has ever been reported where a patient who has never left South Africa, has been shown to harbour *Schistosoma mansoni.*"

There must have been many occasions on which *Schistosoma mansoni* had been brought into the country, but it would appear never to have been able to establish itself.

At last in January, 1920, Annie Porter[19] reported the existence of *Cercariæ mansoni* in molluscs collected from pools in Mayfield and Sydenham, two small villages on the outskirts of Durban, inhabited by East Indians.

In September of the same year Faust[20] reported the presence of the same parasite in material sent to him by Cawston, also from Sydenham, and in June, 1921,[21] Cawston found a boy from this village with eggs both of *Schistosoma hæmatobium* and of *Schistosoma mansoni* in his urine.

It would seem that after many years *Schistosoma mansoni* has at last obtained a footing in South Africa, but only in one small area with an alien population. It will be interesting to see if this parasite will be able to maintain its hold or if it will die out in the course of the next few years.

Conclusions.

1. Australia, lying within the bilharzia belt, is undoubtedly open to invasion by schistosomiasis.

2. Many cases of schistosomiasis have been introduced into the country and are there at the present time.

3. The human schistosomes show great power of adaptability to new molluscan hosts and it is almost certain that some of the fresh water molluscs indigenous in Australia will be found suitable for the development of these parasites.

4. There would appear to be some as yet undefined power which regulates the distribution of endoparasites, permitting their propagation in one place and inhibiting it in another, even where all the conditions would appear on the surface to be favourable to the parasite.

5. Schistosomes, on introduction in sufficient numbers to a country where this inhibitive force appears to act, may obtain a temporary hold and may breed up to a certain point, but, after a time, possibly at the period at which new infective material ceases to be introduced, they die off and the disease disappears.

6. As sporadic cases of schistosomiasis are known to have occurred in Australia after the South African War and as new cases ceased to appear after a short time, it is probable that this inhibitive force exists in Australia and that, though sporadic cases may occur as the result of the late war, the disease will fail to get a permanent hold on the country.

7. This pious hope should not be allowed to stand in the way of all possible measures being taken to trace existing cases of schistosomiasis and to get them to submit to radical treatment.

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Reports of Cases.

HENOCH'S PURPURA.[1]

BY R. J. MILLARD, C.M.G., C.B.E., M.D., CH.M. (SYD.), D.P.H. (CAMBRIDGE),

Medical Superintendent, Coast Hospital, New South Wales.

THE following report of an acute abdominal condition diagnosed as Henoch's purpura is of interest:

C.S., male, aged sixteen years, shop assistant, was admitted to the Coast Hospital on August 18, 1922, complaining of severe pain in the abdomen and vomiting. The patient had been taken ill on August 16 with an attack of pain in the abdomen. This was succeeded by vomiting and the patient felt so ill that he went to bed. On the seventeenth he noticed a rash on the trunk and on the elbows. On admission to hospital abdominal pain was severe and vomiting was continuous. The patient lay with his knees drawn up. Considerable tenderness of the abdomen was present, but not much rigidity. A scattered purpuric rash was present on the trunk and elbows.

On August 18 under general anæsthesia the abdomen was opened over McBurney's point. Abundant free sanguineous fluid was found in the peritoneal cavity. The caecum was found to be intensely inflamed. The appendix was adherent and retro-caecal, but not inflamed. An incision was made in the middle line above the umbilicus. The pancreas and gall bladder were normal. The small intestine generally was in the same dark and congested condition as the caecum. There was no sign of mesenteric thrombosis. Drainage was established through the original incision. Pain and vomiting ceased after the operation and recovery has been uneventful, except for the development of varicella on August 21.

Henoch's purpura or *purpura abdominalis* was first described by Henoch in 1864. It is characterized by recurrent attacks of purpura, with crises of abdominal pain often accompanied by vomiting and sometimes by diarrhœa. Arthritis is frequently present and nephritis is a serious and common complication. Osler has described similar manifestations in erythema and urticaria. It is closely allied to these conditions and is really a member of the angio-neurotic group. It is a disease of early life and is more common in males than in females. The onset varies. It may be acute or come on gradually with a feeling of malaise and headache. Fever is usually, but not invariably, absent. The pain is agonizing and constant and is generally accompanied by profuse tenderness. Although diarrhœa is frequently met with, there may be constipation and a condition of acute intestinal obstruction may be simulated. The prognosis is generally good and more favourable in children than in adults. Osler held that the pain may be due to localized urticarial swelling in the gastro-intestinal wall.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 8, 1922.

Reviews.

THE APPEARANCE OF SYPHILITIC LESIONS.

PROFESSOR VON ZUMBUSCH'S "Atlas of Syphilis"[2] is magnificently set out; the binding is good, the paper of the highest class, the printing excellent and the price (30s.) extraordinarily low.

According to the publishers' preface, the methods of obtaining the illustrations is a new departure in medical literature in that they are reproductions of "coloured photographs, taken direct from the patients, placed at the disposal of the profession, for the first time."

However obtained, the results are magnificent and the plates are even better than those of Jacobi's "Atlas," which was thought to be perfection. The pictures are, as the publisher claims, "true to life and supersede anything achieved in accuracy of coloration."

The book is published for students and general practitioners and displays the common manifestations of syphilis with extraordinary faithfulness.

Its value is incalculable in the way of keeping a practitioner not in daily contact with cases of syphilis familiar with its common lesions.

In addition to illustrations of skin and mucous membrane lesions in the primary, secondary, tertiary and congenital stages of syphilis there are excellent colour reproductions of the microscopical appearance of the *Spirochæta pallida*, stained and unstained, in fluid and in tissue, and of gross pathological appearance of syphilitic cirrhosis, gummata of the liver and testicle, syphilitic aortitis, aortic aneurysm and syphilitic disease of the bones. There are also displayed the typical appearance of the salvarsan and mercurial rashes.

It is impossible to select one illustration as being better than the others in depicting its particular variety of syphilitic lesion, but as instances of accuracy of detail and reproductions in colour of features, skin texture, light and shade Plate 27, showing two syphilitic faces, is inimitable.

This publication is one which everybody in general or special practice would do well to study carefully in detail, so as to form an accurate visual memory of its reproductions and to keep for actual reference when a doubtful case occurs.

[2] "Atlas of Syphilis," by Professor Leo von Zumbusch, Munich; 1922. London: John Bale, Sons & Danielsson, Limited; Demy 4to., with sixty-three illustrations taken direct from Nature by colour photography on heavy art paper and one single colour illustration. Price: 30s. net.

THE TREATMENT OF SYPHILIS.

DR. REGINALD HAYES has recently published the fourth edition of his book on "The Intensive Treatment of Syphilis and Locomotor Ataxia."[1] In this small book of ninety-five pages the author states the well-known fact that inunction is by far the best method of using mercury, but points out that it cannot have its maximum effect unless it is properly carried out. To this end he advocates the Aachen method, which consists of the inunction of a 33.3% mercury ointment by the bare hands of a skilled rubber under proper medical supervision and in addition the use of sulphur water internally and externally. The book contains illustrations showing the sites and method of the rubbing and the formula of the ointment used. It is stated that mercury in the body is formed into an insoluble albuminate and the sulphur water is used because the sulphuretted hydrogen unites with the mercury, making it an active soluble compound.

At Aachen the patients are put through an exhaustive physical examination before treatment is commenced, a practice that should be and is not the routine at other places and clinics. Particular attention is given to the teeth; all old stumps are removed. With general oral hygiene they have little or no gingivitis, even after as many as two hundred consecutive daily rubbings.

For tabetics, in whom any pain or even the thought of pain or discomfort should be avoided, the inunction method of giving mercury is the treatment *par excellence*. A point claimed in its favour is that under the excuse of massage the treatment can be kept secret.

The author concludes with a number of illustrative cases.

It is quite pleasing to learn of an author of a modern book pushing the case for mercury in the treatment of syphilis, as nowadays there is a tendency for many to rely too much on the use of more arsenic and less mercury. At the same time the author gives some notes on the use of arsenic and advocates its use as well.

[1] "The Intensive Treatment of Syphilis and Locomotor Ataxia by Aachen Methods (with Notes on Salvarsan)," by Reginald Hayes, M.R.C.S., etc.; Fourth Edition, Revised; 1922. London: Baillière, Tindall & Cox; Crown 8vo., pp. 99, with four plates. Price: 4s. 6d. net.

The Medical Journal of Australia

SATURDAY, OCTOBER 14, 1922.

The Congress.

The work of preparation for the First Session of the Australasian Medical Congress (British Medical Association) is well under way. A full year before the Session begins the programme of the organization of the Congress and of its sectional and administrative committees has been completed. From the document which appears in another part of this issue it will be seen that the Executive Committee and the able Secretaries have evolved a sound scheme and have progressed far in contriving the details. Several prominent medical practitioners in the United Kingdom, in other parts of the Empire and in the United States of America have been invited to attend as honorary members. Among them the President of the British Medical Association, Professor C. J. Martin, Professor Sir Arthur Keith, Sir Harold Stiles, Sir Robert Jones, Sir John Bland Sutton, Dr. Victor Heiser, Dr. Harvey Cushing and Dr. George W. Crile may be mentioned.

The new organization, like the old, stands under vice-regal patronage, but the Executive Committee and the Federal Committee have determined that Congress must enjoy a greater degree of independence than formerly and that its character shall be strictly scientific. As this new undertaking is the realization of a desire expressed and felt for many years, it is important that the members of the Branches of the British Medical Association in Australia and New Zealand should give it the fullest measure of support.

In the year 1920 we published an historical survey of the Intercolonial (later Australasian) Medical Congress from the time of its initiation in Adelaide in 1886. From this account it appears that, although the Congresses had served a highly useful purpose during the course of thirty-seven years, the need for a more stable constitution, for greater security of the institution and for a stronger link with the body representative of the medical profession in regard to medico-political interests had become increasingly evident. The new Congress has been planned in such a manner that it will be eminently suited for the important task of furthering scientific knowledge, while political matters affecting the profession are left in the hands of the Federal Committee, whose machinery has been created for that purpose. The amount of original work has increased very largely during the course of the last few years in the Commonwealth and in the Dominion. A biennial or triennial congress, if well managed and fittingly supported, cannot fail to achieve much without interfering with the scientific work of the Branches. It provides the members with a common ground for debate at regular intervals. The country member is but rarely in a position to attend meetings of his Branch and consequently these larger and more important meetings will offer to him an opportunity of listening to the leaders of medical thought and of forming a sounder judgement concerning their doctrines than he can do by reading their writings. The early preparations of the Executive Committee give all medical practitioners ample time to make their arrangements for attending the Session of Congress in Melbourne next year.

Attention is directed of all medical practitioners interested in this undertaking to the regulations governing membership. These regulations were published in The Medical Journal of Australia of February 25, 1922. In the first place, every member of any Branch of the British Medical Association in Australasia has the right to enrol as a member on making an application in writing to the Executive Committee and forwarding the prescribed fee, which has been fixed for the First Session at two guineas. Legally qualified medical practitioners who are not members of the British Medical Association, may be admitted to membership of Congress on the following conditions. In the first place, the practitioner must secure nomination by two members of the British Medical Association and this nomination must be published in The Medical Journal of Australia. He must then forward his application to the Executive Committee,

together with the prescribed fee for membership. He must also satisfy the Executive Committee that he is not ineligible for membership of any Branch of the British Medical Association and that he has reasonable grounds for not applying for membership of that association. The Executive Committee will then communicate with the Council of the Branch of the British Medical Association in whose area he resides, to the effect that he has applied for election as a member of Congress. If no valid objection be raised, the Executive Committee may proceed with his election. It will be seen that the method of joining the Session is essentially different from that of the previous Australasian Medical Congress and that no undue hardships are imposed on medical practitioners who are not members of the Branches of the British Medical Association.

The success of the Congress will depend not only on the attendance of medical practitioners, but also on the presentation of scientific communications. The *motif* of the Session will be prevention. This means that clinicians, investigators, pathologists and epidemiologists are invited to put forward original work directly or indirectly connected with the prevention of disease, with the preservation of mass or community health or with the amelioration of the effects of noxes which inflict damage on the people of Australasia. It may be assumed that papers on other subjects will be admitted and that the Executive Committee or the officers of the sections will not refuse proffered communications of good quality not connected with the prevention of disease. Twelve months is not too long a time for the preparation of papers or addresses to be read or delivered before a critical congress audience. Those who have opportunity and ability to study important medical problems, particularly in relation to preventive medicine, are recommended to lose no time in getting to work. The Executive Committee has set an excellent example.

THE BASAL METABOLISM OF PREMATURE INFANTS.

Attention has repeatedly been called in these columns to the common habit among clinicians of building up methods of dealing with infants without regard to the fate of the chemical substances poured into the baby's stomach. The management of infants is admittedly an exceedingly difficult problem requiring experience and a keen perception of physiological processes. As a rule the management of the healthy infant is undertaken with but little guidance by the mother. It is true that the maternity nurse has the best opportunity of instilling first principles into the mother's consciousness at an early stage. In practice these lessons are frequently not delivered with sufficient clearness or force to guarantee a safe sojourn in the nursery for the little one. When as a result of bad management or of a special difficulty caused by an inherent disability of the infant, natural nursing is relinquished in favour of an unnatural method, the mother is usually guided by well-meaning but unphysiological neighbours. The medical practitioner is usually consulted when much harm has been inflicted and when the problem has become unnecessarily difficult. If the advice tendered by the medical practitioner is based on some empirical formula, instead of precise knowledge gleaned from measurements and analyses, the prospects for the infant are not improved. Some suggestive information has been collected from time to time concerning the metabolism of normal infants, although much more is necessary before the actual process of digestion and metabolism can be followed.

Drs. F. B. Talbot, W. R. Sisson, M. E. Moriarty and A. J. Dalrymple have initiated a series of studies into the basal metabolism of premature infants. They have recently published their observations on the relation of basal metabolism to the caloric intake and to the weight curve.[1] The infants were apparently healthy, but as a result of premature birth had a high pulse rate; their temperature mechanism was unstable and they were too weak to suckle at the breast. Some of them were given diluted cows' milk as well as human milk when the mother's supply was deficient. The placental blood was tested by the Wassermann complement fixation method. In no instance did the test yield evidence of syphilitic infection. The metabolism measurements were carried out by Benedict's and Talbot's method with some modifications. The first baby was under observation from the fourth day of life. Its weight decreased at first from 1,600 grammes to 1,350 grammes in the first week of life. The weight curve rose from the eighth day with two remissions until the end of the period, namely, the thirty-third day, when it was 1,800 grammes. On the fourth to the sixth day the basal metabolism was from 45 to 56 calories, while the caloric intake was approximately 120 calories. The second and third babies were two of triplets. The larger infant had a somewhat higher basal metabolism. Like the first baby, they gained in weight when the intake reached 200 calories a day. The fourth and fifth infants were twins. The one started to gain in weight when it was taking 250 calories or 155 calories per kilogram of body weight. The other gained in weight when its diet yielded 200 calories or 155 per kilogram. Here again the caloric intake was between twice and two and a half times the basal metabolism. The sixth infant maintained a

[1] *American Journal of Diseases of Children*, August, 1922.

basal metabolism varying between 80 calories and 150 calories from the tenth to the sixty-fifth days. The weight stabilized and began to increase when the intake represented just under 200 calories in the day. When the basal metabolism reached 150 calories, the weight had increased to 2,500 grammes, or nearly twice the starting weight. The seventh and last infant also showed a gradual increase of the basal metabolism, starting with 75 calories. In seventy days there was an increase of 110% of the basal metabolism, an increase of 190% in the caloric intake and an increase of 41% in weight.

It appears that premature infants require a relatively high caloric value of their diet to bring about a satisfactory increase in weight. Various observers have estimated this at between 110 to 150 calories per kilogram of body weight. In the experiments recorded it was shown that the amount of heat produced by these weakly infants was surprisingly low and was certainly not proportionate to the area of the body's surface. The authors conclude that a relatively large proportion of the food digested by these premature infants goes to form new tissue. The only food completely assimilated was human milk. Under ordinary conditions approximately 10% of the calories supplied are lost in the excreta, but when cows' milk is fed, the loss may reach as much as 20%. A further 10% is required for muscular exercise. It therefore follows that between 70% and 80% of the calories supplied are available for growth.

These figures require control, but if confirmed are of the utmost importance in the determination of the amount of food needed by premature, normal babies. It must, however, be pointed out that the data supplied have no significance in regard to abnormal infants.

CHOLECYSTOTOMY AND CHOLECYSTECTOMY.

It is probably correct to say that in no part of the abdomen has surgical treatment been more disappointing in regard to recurrences than in the gall bladder. The operation of cholecystotomy has been undertaken most frequently for the relief of cholecystitis accompanied by gall stones. After the gall bladder has been drained for a varying period of time, the necessary fistula has been allowed to close and in the majority of instances the patient has suffered no further symptoms and has to all intents and purposes been cured of his disease. The percentage of recurrences of symptoms, however, has always been moderately high. This is not to be wondered at when the causation of gall stone formation is taken into account. The ultimate origin of gall stones from bacterial infection is generally accepted. It is held that clumping of bacteria takes place in the cavity of the gall bladder or in its mucosa and that a protective covering of cholesterin is deposited around them. Moynihan summed up the situation when he stated several years ago that every gall stone was a tombstone erected to the memory of the organisms buried within it. These buried organisms may retain their virulence. Thus it is recorded that a patient who had suffered from enteric fever at the age of eight years, was operated on for gall stones at the age of forty-three and that living typhoid bacilli were recovered from the interior of the stones. Brown in 1919 made cultures of seventy gall bladders and succeeded in growing streptococci from 75% of those in which extensive pathological changes were present. It is also necessary to remember that the presence of gall stones in the gall bladder is productive of pathological conditions, both in the gall bladder itself and in the neighbouring parts. When gall stones are removed, the advance of these secondary conditions is probably checked and their effects mitigated, but the original site of gall bladder infection in the mucosal or muscular layers of that organ is left in a potentially active condition. It is for this reason that cholecystectomy has been used in an increasing number of cases.

The subject has been recently discussed by Mr. James Sherren.[1] Mr. Sherren states that ten years ago in one hundred consecutive operations for gall stones he had thought cholecystectomy necessary in twenty-nine instances and that in his last hundred operations he had performed cholecystectomy primarily on ninety-four patients. The remaining six patients suffered from acute conditions in which primary cholecystecomy was deemed inadvisable, and in two the gall bladder was removed at a second operation three weeks later. In a series of one hundred and eighty-four cholecystectomy operations performed over three years ago for stones confined to the gall bladder there were six deaths, three of which were due to pulmonary complications. None of the surviving patients suffered any recurrence. In a series of one hundred and fifty-two patients subjected to cholecystotomy during the same period twenty-nine suffered recurrences and five others died within four years of carcinoma of the gall bladder.

Mr. Sherren states that, though external examination of the gall bladder failed to show anything abnormal, cholecystectomy often revealed not only small calculi, but crystals of cholesterin and tiny calculi embedded in the mucosa. He also discovered chronic cholecystitis of the well-known "strawberry" type and the typical thickening at the fundus known as adenoma. He frequently met with this adenomatous structure associated with gall stones and in two instances, though the appearances were not unusual, microscopical section showed the presence of columnar celled carcinoma. It is interesting to note that Erdmann in a series of two hundred and twenty-four patients operated on for disease of the gall bladder found primary carcinoma in fifteen, or 6.7%. Mr. Sherren concludes that the skilled surgeon at the present time must justify his choice of cholecystotomy and not that of cholecystectomy.

In considering this question on the evidence offered by Mr. Sherren no other conclusion can be formed.

[1] *The British Journal of Surgery*, July, 1922.

Australasian Medical Congress (British Medical Association).

ORGANIZATION OF CONGRESS.

THE FIRST SESSION OF THE AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION) will be held from Monday, November 12, 1923, to Saturday, November 17, 1923 (both days inclusive) in the buildings of the University of Melbourne.

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General Information.

Work of Congress.

It has been resolved by the Executive Committee that:

In all the work of the Congress particular attention shall be given to the preventive aspect, including medical research.

In order to carry out the intention of this resolution, it is expected that in all papers and discussions particular attention shall be given to the preventive aspect of the subject treated.

The attention of members of the Australian and New Zealand Branches of the British Medical Association is drawn to the fact that the Australasian Medical Congress is now controlled by the Federal Committee of the British Medical Association in Australia and that the First Session under this new control is to be held in Melbourne from Monday, November 12, 1923, to Saturday, November 17, 1923, in the buildings of the University of Melbourne.

Subscription.

The subscription for membership of the Congress is two guineas, which should be sent to the Local Secretary for the particular State or Dominion in which the intending member resides. In the State of Victoria subscriptions should be sent to the Honorary Treasurer, Dr. C. H. Mollison, 41, Spring Street, Melbourne.

The attendance and support of the members of the Australian and New Zealand Branches of the Association are cordially invited.

It is hoped that members of the Branches will endeavour to make the First Session memorable from the point of view of the quality of its work and the number of those who attend.

Travelling Facilities.

The Railway Commissioners of the Commonwealth and of the Australian States have agreed to concessions as hereunder stated:

In accordance with the usual practice, delegates travelling from other States to attend the meeting of the Australasian Medical Congress at Melbourne in November, 1923, will be issued single tickets for both the forward and return journeys at two-thirds of the single fare, provided that not less than six delegates travel from a particular State; and the concession will apply to the wife of a delegate if she accompanies her husband.

The tickets will be issued so as to enable the delegates to arrive at their destination not more than three days prior to the commencement of the Congress and to begin the return journey within one month from the date the conference ceases; but the journey cannot be broken except for one day in passing through a capital city.

(Sgd.) J. S. Rees,
Acting Secretary.

Accommodation.

The week immediately preceding the Congress is Melbourne Cup Week, but it is not anticipated that there will be any difficulty in securing accommodation for visitors during Congress Week. In due time the Entertainment Committee will publish information in detail as to the accommodation available.

Constitution.

Members of the Association are referred to the issue of The Medical Journal of Australia of February 25, 1922, for the Constitution and Regulations of the Congress and in especial to Regulation 4 regarding membership and Regulation 13 regarding Sectional Meetings.

Information.

Any information required on any matter concerning the Congress should be sought from the Local Secretary of the State or from the General Secretary of Congress, all of whose addresses are given in the accompanying list of the Organization of Congress.

Notices.

Notices of matters concerning the Congress will appear from time to time in issues of The Medical Journal of Australia.

If there be any errors or omissions in the accompanying list, please notify the General Secretary at once.

It is requested that members of the Association will be good enough to keep this list for reference.

A. L. Kenny,
General Secretary of Congress.

Medical Society Hall,
Brunswick Street South,
East Melbourne, Victoria.

September 15, 1922.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

Optic Nerve and Accessory Sinuses.

J. VAN DER HOEVE (*Archives of Ophthalmology*, May, 1922) discusses the signs and diagnosis of optic nerve disease under the various headings of choked disc, papillitis, atrophy and retro-bulbar neuritis and concludes with a general statement that the ophthalmologist has in the eye no sign at all to distinguish the origin of a retro-bulbar neuritis. Turning to the diagnosis of sinus disease, in spite of the rhinological examination and the information derived from a skiagram, the author makes a second statement that the rhinologist cannot say with absolute certainly that a person has no sinus disease. The anatomical relation between the optic nerves and the sinus varies considerably; the bony wall between the two may be quite thick and sometimes may be absent. The exposed nerve may be affected by contact with pathological secretion or the swelling of the nerve from toxic œdema may cause more disastrous results if the nerve is enclosed in a bony canal. In this case the pressure may be sufficient to cause choked disc or atrophy. They should distinguish reparable optic nerve affections caused by toxins, œdema and pressure from irreparable changes caused by degeneration and atrophy following the same processes, but in stronger degree. When optic nerve disease is associated with known sinus disease, the treatment is obvious; the sinus disease must be attacked. But in the absence of any indication of sinus affection and with no other explanation for the optic nerve disease, it is difficult to determine whether the sinuses should be opened or not. Many such instances have been reported where operation proved of great service. The question must be considered from many points of view and the author thinks that further statistics may throw light on the subject.

Living Sutures in Ptosis Operation.

W. W. WRIGHT (*Archives of Ophthalmology*, March, 1922) describes the use of sutures of *fascia lata* in the operation for ptosis. An incision, twelve and a half centimetres (five inches) in length, is made over the outer side of the thigh and the *fascia lata* exposed and cleaned. Two parallel incisions are then made in the *fascia* four to five millimetres apart and the strip freed from the muscle. Before cutting the extremities it should be split longitudinally to form the two sutures required. They are threaded at both ends on curved needles and fixed firmly with silk. The horn spatula is placed under the upper lid and two small punctured wounds are made through the skin about ten millimetres apart and six millimetres above the lid margin. A transverse incision ten millimetres long is made over the brow. A needle is inserted into one puncture and passed subcutaneously in a transverse direction, to emerge at the other puncture. Each needle is then passed vertically under the skin of the lid, to emerge at each extremity of the brow incision. Two such sutures are inserted in the lid. The ends of the sutures are now drawn on until the lid is raised to the desired heighth and then tied in the transverse wounds. The knot should be reinforced by a catgut ligature. The author adopted this procedure with the lid of a boy. A good degree of motility subsequently resulted.

Ocular Manifestations of Multiple Sclerosis.

BESIDES transitory ocular palsies of nuclear origin in 20% of patients suffering from multiple sclerosis and the almost constant presence of nystagmus, W. A. HILDEN (*Archives of Ophthalomlogy*, March, 1922) describes as of particular interest certain optic nerve symptoms useful in diagnosis and of assistance in relieving the patient of the fear of impending blindness. A central scotoma of rapid onset, if toxic conditions are excluded, must mean either infection from the paranasal sinuses or multiple sclerosis. If the former be excluded by examination and X-ray photography, a diagnosis of multiple sclerosis is justifiable, especially in the presence of additional signs, such as ankle clonus, the Babinski reflex or diminished abdominal reflexes. Characteristic plaques of inflammatory exudation develope in the optic nerve or tract at any point, but with great frequency in the axial fibre bundle. The pressure interferes with vision, but as the axis cylinders are not destroyed, restitution of vision frequently takes place. The disc pallor may cover the whole disc or be confined to the infero-temporal third and this variety is to a certain degree characteristic of multiple sclerosis. Defects of the field of vision usually take the form of central and paracentral scotomata. The visual disturbance may reach its maximum in a few days and recovery may take place in the same time; or the disturbance may continue for years with remissions and relapses. Unlike atrophies of the optic nerve of other origin, this variety never leads to blindness.

Lipæmia Retinalis.

ACCORDING to W. F. HARDY (*Archives of Ophthalmology*, November, 1921) lipæmia usually occurs in diabetes, but it also occurs in pregnancy, chronic alcoholism, diseases of the liver, nephritis, malaria, tuberculosis, cholera and phosphorus poisoning. Fat in normal blood varies from 1‰ to 3.25‰; in *lipæmia retinalis* it varies from 10% to 26%. The author's patient, a white man, aged 29, complained of a skin condition which was diagnosed as xanthoma. He had sugar in his urine and 9.5% of fat in his blood. Eye examination revealed *lipæmia retinalis*. Hale-White maintained that the turbidity was not entirely due to fat globules, but other observers, with better staining reagents, have demonstrated fat droplets of all sizes. The colour of the retinal vessels has been described as salmon; the author describes his as greyish white or like malted milk. The colour depends upon the light used, the amount of fat present and the background. The veins and arteries look alike as flat ribbons twice their normal width. As the patient improved the fundus picture returned to normal in two weeks. *Lipæmia retinalis* produces no pathological changes in the eye, but, of course, the diabetes may. In health the visible part of the blood in the vessels comprises the axial stream, consisting of corpuscles whereon the peripheral part is invisible plasma. In lipæmia the plasma becomes opaque and visible, consequently the vessel appears double its usual calibre.

Advancement Without Incision.

S. L. ZIEGLER (*American Journal of Ophthalmology*, April, 1922) describes his method of capsulo-muscular advancement without incision, by means of a single double-armed thread. The muscle is grasped through the overlying conjunctiva by fixation forceps fifteen millimetres behind the *limbus*. Each needle is passed twice through muscle and conjunctiva, one above and the other below, thus forming a "whipstitch" on the upper and lower margins of the muscle. Each needle is then carried backwards beneath the capsule for ten millimetres, brought through the conjunctiva and then carried forwards and entered vertically through the conjunctiva two millimetres behind the *limbus* and five millimetres below or above the horizontal plane. Each needle should dip into the sclera and emerge two millimetres below or above the horizontal plane, leaving an intermediate space of four millimetres for tying the two ends of the suture. The suture is now tied firmly until the squint is slightly over-corrected. A preliminary tenotomy of the opposing muscle, more or less complete according to requirements, is usually performed.

Intermittent Exophthalmos.

W. G. M. BYER (*Archives of Ophthalmology*, November, 1921) records the history of a patient, a married woman, who complained that on stooping or lying on the right side her right eye bulged forward. The patient was otherwise a healthy woman. The right eye in the upright position was sunken. Following stooping, pressure on the internal jugular, turning the head to the left or to the right, throwing the head well back, holding the breath after forced expiration, the eye bulged to a varying degree, even as much as twenty-five millimetres. There was no limitation of movement of the globe. The fundus was markedly congested and the disc abnormally vascularized. The author concludes that there was a varicose condition of the orbital veins, supposedly of congenital origin; there was inadequate collateral drainage. A skiagram showed the presence of a supernumerary cervical rib, which may have had some bearing on the jugular constriction.

LARYNGOLOGY AND OTOLOGY.

Deaf-Mutism.

J. S. FRASER (*Journal of Laryngology and Otology*, January, February and March, 1922) states that he prefers Hammerschlag's classification of cases of deaf-mutism, namely, those apparently due to errors in development and those due to inflammatory conditions or traumatism, the latter being always acquired, whether in fœtal or post-fœtal life. Deaf-mutism due to errors in development may be congenital or acquired. It is hereditary either directly or indirectly, occurs more than once in the same generation and is associated with other degenerative conditions. Fraser believes that the condition is more often congenital than acquired. Congenital deaf-mutism includes cases of cretinic deafness and of sporadic congenital deafness. In cretinic deafness (Siebenmann's type) there are changes in the middle ear and labyrinth capsules, including the window niches, whereas the inner ear is usually normal. There may be some hearing, but owing to the accompanying feeble mental development the result is deaf-mutism. In sporadic congenital deafness the membranous labyrinth and the eighth cranial nerve are at fault. There are several varieties. Firstly, there is aplasia of the whole labyrinth (Michel's type). Secondly there are cases in which both the bony and membranous labyrinths are affected (Mondini's or Alexander's type). In this type there may be some hearing. There is abnormality of growth, possibly the persistence of a fœtal condition with arrest of development. There may be atrophy of part of the vestibular apparatus. In the third place there is congenital malformation affecting both the (membranous) cochlear and vestibular apparatus. This form is frequently associated with *retinitis pigmentosa*. The vestibular reactions are almost completely absent, there may be a little auditory power and many of the patients are mentally feeble. The fourth class are those distinguished by sacculo-cochlear degeneration (Scheibe's type) and comprise about 70% of the cases of deaf-mutism. In this type the utricle and canals are intact. Hearing remnants are usually present. The vestibule apparently reacts normally to rotation and caloric tests. The saccule and cochlear canal show faulty development. Corti's organ may be absent in parts or unrecognizable. Certain brain changes have been found. The connexion, if any, between congenital deafness and oto-sclerosis is dubious. Deafness due to brain lesions has been but little investigated. Acquired or inflammatory deaf-mutism is due to labyrinthitis in intra-uterine or post-fœtal life. The former is of meningitic origin; the latter may be due to traumatism, *otitis media* or to purulent meningitis. Meningitis is the most frequent cause and may have its origin in scarlet fever, congenital syphilis, osteo-myelitis, mumps and perhaps *purpura hæmorrhagica*. The author reviews the literature of deaf-mutism and states that he favours Itard's clinical classification: Group I.: Patients who can hear conversation voice at two metres. Group II.: Those who can hear the raised voice close to the ear. Group III.: Those who can hear vowel sounds. Group IV.: Patients who can hear loud noises, such as a rattle, trumpet, whistle or hand-clapping. Group V.: Those with total deafness. Alexander states that patients in Group V. can be excluded as early as six months, provided the infants are otherwise mentally normal. Patients in Group III. can only be satisfactorily examined at the age of two years and patients in Groups I. and II. at the age of four to five years. Alexander holds that the totally deaf patients drag their feet, because they cannot hear the unpleasant sound produced. Total bilateral deafness, according to Brock, is usually acquired; 25% of deaf-mutes have some remnants of hearing power. Bezold laid stress on the necessity of segregating those with remains of hearing from the totally deaf. In deaf-mutes with a doubtful history it can be assumed that the condition is congenital if the static labyrinth is excitable; this may also be inferred if remains of hearing are present.

Epidemic Cerebro-Spinal Meningitis.

IAN MCKENZIE (*The Journal of Laryngology and Otology*, August, 1922) states that when the meningococcus is present in the naso-pharynx, there is usually a catarrhal inflammation there. The path followed by the meningococcus in passing from the naso-pharynx to the meninges is unknown. There is no clinical or pathological evidence that the route is through the roof of the nose by way of the olfactory nerves and the cribriform plate. Three other routes are suggested, but none has ever been actually demonstrated. The first is by way of the sphenoidal sinus and bone. The second is by the blood stream producing a general septicæmia of which meningitis is merely one of the manifestations, or, without producing septicæmia, the infection finds in the meninges an environment favourable to its activity. The third suggested route is by way of the Eustachian tube and middle ear to the labyrinth and meninges. The meningococcus has often been observed in suppuration of the middle ear during an attack of cerebro-spinal fever. Deafness in cerebro-spinal fever is, as a rule, bilateral. Recovery, even from absolute deafness, has been recorded, but in this case the deafness was probably due to toxic influences.

Diathermy in Laryngeal Growths.

FRANK J. NOVAK (*The Illinois Medical Journal*, April, 1922) holds that in carcinomata of the larynx in its early stages, before a generalized dissemination of metastases has occurred, diathermy offers advantages over all other known measures. The local application of sufficient heat destroys the tumour mass and a lower degree of heat to the periphery of the tumour mass and beyond its limits causes inhibition of the growth of the migrating neoplastic cells and occlusion of the lymph spaces and channels. Suspension laryngoscopy and chloroform anæsthesia are recommended. A round, flat electrode tip on a shank twenty centimetres long is applied to the tumour, the inactive electrode, a large, wet pad, is applied to the patient's back. The amount of current depends on the size and location of the tumour. One thousand to fifteen hundred unit ampères are generally used and the average length of exposure is twenty seconds. Fractional coagulation is preferable to coagulation of the whole mass at one time. The necrotic mass separates in about ten days and a smooth, hard scar forms in from three to six weeks. Diathermy *plus* radiation is superior to diathermy or radiation alone. A preliminary tracheotomy is imperative.

Alcoholic Injections in Hyperæsthetic Rhinitis.

IN the treatment of simple hydrorrhœa, spasmodic coryza, nasal reflex asthma and hay fever OTTO J. STEIN (*Illinois Medical Journal*, July, 1922) claims good results from the intranasal injection of alcohol into the nasal nerves. After thorough preliminary cocainization he introduces with a straight steel needle nine centimetres in length and of twenty-two to twenty-four gauge 0.6 cubic centimetre (ten minims) of a 75% dilution of alcohol with fresh sterile water into the nasal nerve as it emerges into the antero-superior angle of the nasal chamber a millimetre or two lateral to the septum. He then injects Meckel's ganglion with the same amount through the spheno-palatine formamen, using a needle ten centimetres long, with sixteen to eighteen gauge, bent at a right angle ten millimetres from the point. A longer or shorter bend may be required in wide or narrow nasal fossæ. The needle is gently hooked behind the middle turbinate just below its posterior attachment and the point thrust outwards. Reinjections are required in seasonal cases. Prolonged pain following the injections may require the use of morphine.

Facio-Hypo-Glossal Anastomosis.

LIONEL COLLEDGE (*The Proceedings of the Royal Society of Medicine*, August, 1922) records the case of a girl, aged sixteen, with right-sided facial paralysis persisting six months after operation on the mastoid and labyrinth. Reaction of degeneration was present. The hypo-glossal nerve was divided and the central end anastomosed to the peripheral end of the facial. The peripheral end of the hypo-glossal was anastomosed to a slip from the spinal accessory. Six months later tone had returned to the facial muscles at rest and movements of the tongue were associated with movements of the right half of the face, but there was dissociated voluntary control of that side of the face.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Queensland Branch of the British Medical Association was held at the B.M.A. Building, Adelaide Street, Brisbane, on September 1, 1922, DR. G. P. DIXON, C.B.E., the President, in the chair.

Foreign Body in the Trachea.

DR. WALTER CROSSE exhibited a hairpin removed from a girl, aged three years, by means of Jackson's bronchoscope. The hairpin had been arrested at the bifurcation of the trachea.

Hydro-Nephrosis.

DR. E. S. MEYERS showed pyelograms of hydro-nephrosis and renal calculi.

Aneurysm of the Innominate Artery.

DR. VAL. MCDOWALL showed a skiagram of an aneurysm of the innominate artery.

Urinary Infection.

DR. A. S. ROE read a paper entitled "Urinary Infection" (see page 433).

DR. S. F. MACDONALD read notes on the medical aspect of urinary infections (see page 435).

DR. D. GIFFORD CROLL, C.B.E., read notes on renal infections in children (see page 436).

DR. J. V. DUHIG read notes on the ætiology of urinary infections (see page 437).

DR. G. P. DIXON, speaking as a surgeon, stated that renal infections could be divided into two classes: (*i.*) Those which were associated with casts and albumin in the urine, as in Bright's disease, or with an excretion of toxin from a focus, and (*ii.*) those which were associated with pus and bacilli in the urine. In the second class the bacilli, during their passage through the kidney, damaged this organ. He emphasized the necessity of a thorough and complete examination in every case of suspected disease of the urinary system, in order to avoid many unpleasant surprises. The examination of the urine should be repeated at frequent intervals and should include the urea concentration test. Skiagrams of both kidneys, ureters and bladder should be prepared and cystoscopical examination of the bladder should be carried out. In addition, the various dye tests should be used. At times ureteral catheterization, with the use of the opaque catheter and a subsequent X-ray examination, was necessary. When pus and bacilli were found in the urine, the cause of obstruction, such as stone, enlarged prostate or kinking of the ureter, should be removed. If no gross cause could be discovered, he did not share the optimism of Dr. Roe regarding treatment. He had had satisfactorily good results from injections of collargol. It caused no inconvenience nor pain and did not damage the renal tract. He had seen little good from the usual treatment by massage of chronic prostatis. He asked Dr. Roe what treatment he adopted in non-obstructive infections in which pyuria and bacilluria were present.

DR. R. A. MACLEOD could not understand how fatigue could cause damage to the kidney. This was not seen with nerve or muscle fatigue. In the treatment of pyelitis in children he advocated the exhibition of alkalies every two hours during the day and twice during the night until the urine became alkaline. The common mistake was to omit the administration of the drug during the night-time. Three and a half grammes (one drachm) of potassium citrate was supposed to render the urine alkaline, but it generally took six times this dose to secure this effect. While some writers advocated large doses, he found that they usually made the patient sick. He preferred to give 1.8 grammes every two hours. Still had found abscesses in the interstitial portions of the kidney and not in the tubules in three patients after death. Alkalies could not reach this situation. Tuberculosis must be excluded in all instances of pyelitis. It was often difficult to find tubercle bacilli in the urine. He had occasionally obtained good results from sandalwood oil and "Urotropin." He had been surprised when carrying out routine examination of the urine of hospital patients at the prevalence of albuminuria and bacilluria. The presence of albumin in the urine was not such a bogey, if the functional renal tests were satisfactory.

DR. E. S. MEYERS said that during fatigue acid bodies were reduced and there was an increase in the hydrogen-ion concentration. This threw an extra load on the kidneys and allowed bacteria to enter and obtain a foothold. *Bacillus coli communis* might be a secondary invader and the primary cause. More information might be obtained by the study of focal infections.

DR. A. GRAHAM BUTLER, D.S.O., welcomed Dr. Roe's paper, chiefly because he had put forward a theory based on ideas of the ætiology of renal disease. A working hypothesis of the diseases of the kidney was required more urgently than of those of any other organ of the body. It must be based on physiological facts. An immense mass of facts had been accumulated in connexion with renal disorders, but the integration of these facts into a comprehensive system of ætiology had scarcely commenced. Prevention was therefore handly thought of. The distinction between medical and non-medical diseases of the kidneys was drawn as a result of ignorance of exact causation rather than as a result of a reasoned ætiological classification. Infective diseases of the kidneys, from bacilluria onward, were thus marked off from Bright's disease. Yet the most important ætiological factors in Bright's disease of which they had definite knowledge, were infective. The kidney was among the most primitive organs and its functions among the fundamental manifestations of life. The first terrestial forms of life brought with them from the sea an organ, whose function was to maintain the saline equilibrium of the body fluids. This organ had developed into the modern kidney, whose chief function was the maintenance of the saline tension of the body fluids. It was interesting to note that the body fluids had the same density as the sea, not as it was at present, but as it had been at the beginning of the terrestrial life of the globe. The function of the kidneys and the allied function of the maintenance of the hydrogen-ion concentration were of a very delicate and complicated nature. The disorders of the kidney were correspondingly difficult to understand and had to be studied in a different way to the cruder conditions, such as gross infections. But, as in the more highly differentiated disorders of the functions of the brain, the ultimate basis of study must be the processes of metabolism and of toxæmia and infection, coordinated with precise knowledge of structure. Classification and research, based on other circumstances of the diseases, must be preliminary and tentative. This was often ignored. He thought that Dr. Roe's hypothesis of excessive metabolism was reasonable. It would be difficult otherwise to understand how chemical substances circulating in the blood, such as lead, toxin, etc., damaged the kidney. It would assist and direct the integration of their clinical experience of renal disorders and would help the solution of the problem. Renal disease was probably the greatest source of unexpected tragedies and hopeless disorders. Their possibilities of preventing these diseases or of caring for the patients were at present very limited.

DR. T. H. R. MATHEWSON noted that Dr. Roe had emphasized the importance of fatigue in the development of pyelitis. Mental stress was a very important cause of fatigue. Worry and other psychic factors upset the balance of the body. He considered that they isolated the functions of the body too much.

In his reply DR. ROE thanked the members for their reception of his paper. He stated that his hypothesis was of value from a clinical point of view. The treatment of chronic pyelitis frequently yielded depressing results. The attacks often occurred in cycles and consequently the urine should be tested at regular intervals and its reaction should be kept alkaline. Chronic pyelitis in females often underwent exacerbations during the pre-menstrual week. He insisted in the avoidance of fatigue, kept the bowels open, limited the diet and rendered the urine alkaline. He held the opinion that no patient over forty years of age was cured of chronic pyelitis. The majority of patients with eclampsia had a history of renal trouble at some period. Acidosis was not the only cause of bacterial infection, but it rendered a person liable to such infections.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

THOMPSON, GEORGE SYDNEY, M.B., Ch.M., 1922 (Univ. Sydney), Royal Prince Alfred Hospital, Sydney.

THE undermentioned have been elected members of the Victorian Branch of the British Medical Association:

BACKWELL, CLAUDE EWART, M.B., B.S., 1922 (Univ. Melbourne), 34, Myers Street, Geelong.

GILBERT, DUNCAN THOMAS, M.B., B.S., 1922 (Univ. Melbourne), 58, Fellows Street, Kew.

SPEED, ASHCROFT DERRINALL, M.B., B.S., 1922 (Univ. Melbourne), 24, Lewisham Road, Windsor.

SPENCE, ROBERT ALLAN, M.B., B.S., 1922 (Univ. Melbourne), 6, Bond Street, South Yarra.

WHITE, FREDERICK JOHN, M.B., B.S., 1922 (Univ. Melbourne), 81, Tooroonga Road, East Malvern.

WORCH, HAROLD CHISHOLM, M.B., B.S., 1922 (Univ. Melbourne), 61, Stanthorpe Street, Malvern.

TESTIMONIAL TO PROFESSOR SIR HARRY ALLEN.

A REPRESENTATIVE meeting of members of the medical profession was held in the Medical Society Hall, East Melbourne, on September 21, 1922, for the purpose of considering a proposal to establish a fund to be called the Professor Sir Harry Allen Testimonial Fund. MR. F. D. BIRD, C.B., was in the chair.

MR. BIRD explained that the suggestion to give a definite recognition to the eminent services rendered by Professor Sir Harry Allen had originated with the Melbourne Medical Association. This body had forwarded the suggestion to the Victorian Branch of the British Medical Association and to the Victorian Medical Women's Society and had asked for their cooperation. Both these bodies had expressed their hearty approval. A provisional committee of the three societies had been formed and he, the speaker, had been elected chairman. DR. C. H. MOLLISON had been elected Honorary Treasurer and DR. W. G. DISMORE UPJOHN and DR. B. T. ZWAR Honorary Secretaries.

Subscriptions aggregating about £190 and ranging from one guinea to fifty pounds had been received. Promises of support had been obtained from London through DR. J. W. DUNBAR HOOPER, in Sydney from PROFESSOR H. G. CHAPMAN, in Adelaide from DR. C. T. C. DE CRESPIGNY, in Bendigo from DR. W. J. LONG, in Ballaarat from DR. E. CHAMPION and DR. W. A. SPRING, in Sale from DR. G. A. HAGENAUER and in other centres.

It was intended to carry out the presentation at the time of the First Session of the Australasian Medical Congress (British Medical Association) in Melbourne in November, 1923.

The following medical practitioners present at the meeting were appointed to a general committee, with power to add to their number: G. ROTHWELL W. ADAM, C. A. ALTMANN, A. V. M. ANDERSON, J. H. ANDERSON, C. BAGE, F. D. BIRD, DOUGAN BIRD, L. J. BALFOUR, SIR JAMES BARRETT, W. R. BOYD, J. S. BUCHANAN, S. O. COWEN, F. L. DAVIES, H. B. DEVINE, CONSTANCE ELLIS, P. W. FARMER, R. H. FETHERSTON, F. W. GREEN, T. E. VICTOR HURLEY, J. LEON JONA, L. S. LATHAM, A. FAY MACLURE, E. ALAN MACKAY, G. A. D. MCARTHUR, C. H. MOLLISON, W. H. J. MOORE, J. NEWMAN MORRIS, R. H. MORRISON, H. ALAN S. NEWTON, W. S. NEWTON, W. F. ORR, W. J. PENFOLD, C. PERRY, ISABELLA A. PHILLIPS, E. E. R. SAWREY, M. D. SILBERBERG, E. T. W. SINCLAIR, R. R. STAWELL, G. A. SYME, W. G. DISMORE UPJOHN, J. RAMSAY WEBB, P. S. WEBSTER, R. R. WETTENHALL, A. E. ROWDEN WHITE, J. WILLIAMS, A. JEFFREYS WOOD, W. A. WOOD and B. T. ZWAR.

The following were appointed members of an executive committee, with power to add to their number: DRS. F. D. BIRD, A. V. M. ANDERSON, F. L. DAVIES, H. B. DEVINE, CONSTANCE ELLIS, J. GORDON, J. W. DUNBAR HOOPER, C. H. MOLLISON, W. S. NEWTON, G. A. SYME, W. G. DISMORE UPJOHN, J. RAMSAY WEBB and B. T. ZWAR.

Obituary.

CYRIL LOWTHER CLARKE.

IN our issue of September 23, 1922, we recorded the fact that Cyril Lowther Clarke had been killed in a motor car accident at Peterborough, South Australia, on September 16. He was returning from some races with four youngsters when one of the tires collapsed, with the result that the car was overturned. Both Lowther Clarke and a boy, aged thirteen years, were fatally injured, while the others escaped with more or less severe injuries. Despite the skilled attention of his colleagues, he died a short time later. The tragedy has evoked very wide-spread regret, for he had earned great popularity by his sterling worth, his manliness and his fearlessness.

Cyril Lowther Clarke was born at York in 1884. He was the son of the Reverend H. Lowther Clarke, afterwards Archbishop of Melbourne. His early school days were spent in Oxford and later he entered the Bedford Grammar School. At the age of eighteen his father entered his episcopal duties in Melbourne and took his family with him to Australia. Cyril Lowther Clarke entered the Medical School at the University of Melbourne, where he became a distinguished student. In 1908 he graduated in medicine and surgery with honours. Early in 1909 he was appointed resident medical officer at the Kyneton Hospital, Victoria. Later he practised in the Kalgoorlie district, in Western Australia and still later he re-visited England for a short period. In April, 1911, he settled in Peterborough, South Australia. He soon gained the esteem and confidence of the community and the affection of his colleagues. He was known as a sound practitioner, a man of considerable ability and inexhaustible energy and an earnest citizen. He gained the appointments of Medical Officer of Health for the Corporation of Peterborough and of medical referee for pensions under the Commonwealth Government. In November, 1911, he joined the Australian Army Medical Corps Reserve with the commission of Captain. Early in 1915 he offered himself for service with the Australian Imperial Force and on August 1, 1916, he obtained his commission as Major. In 1917 he was attached to the Fourteenth Australian General Hospital and he remained in this position, rendering excellent service, for a considerable time. In 1918 he was transferred to the Fifth Light Horse Field Ambulance. On January 13, 1919, he was promoted to the rank of temporary Lieutenant-Colonel and for a time he served as Assistant Director of Medical Services with the Australian Mounted Division. He returned to Australia in December, 1919.

On resuming his practice, he was greeted with a cordial welcome from all classes and he had the gratification of learning that his admirable qualities had not been forgotten during the years of absence. In 1920 he entered civic duties and was elected Chairman of Finance of the Municipal Council. In December, 1921, he was persuaded by his admirers to stand for election as Mayor of Peterborough and in due course he was successful. His activity in this respect is said to have been very considerable and municipal matters were greatly improved under his wise and capable administration.

He was President of the local branch of the Returned Soldiers' Association and was always ready to help a returned man in need. Indeed, it would seem that his kindness of heart and his sympathy with those not well endowed with this world's goods interfered not inconsiderably with his own financial prosperity. He was less commercial in his methods than the majority of his compeers and his generosity and broad-mindedness were proverbial.

He leaves a widow and two sons, who have received from near and far testimony of the affection and regard in which he was held by the citizens, by his patients and by all his colleagues. His life was made up of useful service and valuable aid rendered with an unusual, bright cheerfulness. He will long be remembered.

Naval and Military.

APPOINTMENTS.

THE following appointments have been promulgated in the *Commonwealth of Australia Gazette*, Nos. 77 and 78, of September 21 and 28, 1922:

Royal Australian Naval Reserve.

EDWIN CHARLES EAST, F.R.C.S., L.R.C.P., M.R.C.S., is appointed Surgeon-Lieutenant, dated 1st May, 1922.

Australian Military Forces.

THIRD MILITARY DISTRICT.

Australian Army Medical Corps.

CAPTAIN J. C. P. STRACHAN is transferred to the Reserve of Officers and to be Major, 1st July, 1922.

SIXTH MILITARY DISTRICT.

Australian Army Medical Corps.

LIEUTENANT-COLONEL H. N. BUTLER, D.S.O., to be supernumerary to the establishment of Lieutenant-Colonels, with pay and allowances of Major, 1st July, 1922.

MAJOR AND BREVET LIEUTENANT-COLONEL W. L. CROWTHER, D.S.O., to be supernumerary to the establishment of Majors, with pay and allowance of Captain, 1st July, 1922.

COMPARATIVE ANATOMY OF THE HUMAN BRAIN.

IT is announced that DR. W. COLIN MACKENZIE will deliver a lecture on "Recent Researches on the Comparative Anatomy of the Human Brain, with Special Reference to the Reptiles and Mammals of Australia," in the upper lecture room at the Royal Society's Hall, Melbourne, on October 16, 1922, at 8.15 p.m.. The lecture will be illustrated by original diagrams, macroscopical and microscopical specimens and lantern slides from the Museum of the Institute of Anatomical Research.

Medical Appointments.

IT is announced that the probationary periods of the appointments of the following medical officers of the Commonwealth Department of Health have been extended for six months from September 10, 1922: DR. F. T. WHEATLAND (B.M.A.), DR. K. R. MOORE (B.M.A.), G. M. HEYDON (B.M.A.) and R. D. MCINTOSH (B.M.A.).

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DR. J. BROWN (B.M.A.) has resigned his position as medical officer in the Laboratories Division of the Commonwealth Department of Health.

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DR. E. CUTHBERT HALL (B.M.A.) has been appointed Government Medical Officer at Parramatta, New South Wales.

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DR. J. E. O'SULLIVAN (B.M.A.) has been appointed Government Medical Officer at Blair Athol, Queensland.

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DR. ALAN DAVISON (B.M.A.) has been appointed Government Medical Officer at Cooroy, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

RENWICK HOSPITAL FOR INFANTS: Honorary Ophthalmic Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Societies Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B. M. A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

OCT. 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
OCT. 18.—Western Australian Branch, B.M.A.: Branch.
OCT. 20.—New South Wales Branch, B.M.A.: Section of Pædiatrics.
OCT. 21.—Northern Suburbs Medical Association, New South Wales.
OCT. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
OCT. 25.—Victorian Branch, B.M.A.: Council.
OCT. 26.—South Australian Branch, B.M.A.: Branch.
OCT. 26.—Western Medical Association (Orange), New South Wales.
OCT. 26.—Brisbane Hospital for Sick Children: Clinical Meeting.
OCT. 27.—New South Wales Branch, B.M.A.: Branch.
OCT. 27.—Queensland Branch, B.M.A.: Council.
OCT. 30.—Victorian Branch, B.M.A.: Council Nomination Paper Issued.
NOV. 1.—Victorian Branch, B.M.A.: Branch.
NOV. 3.—Queensland Branch, B.M.A.: Branch.
NOV. 8.—Western Australian Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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